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ALTERNATING PLEURISY WITH EFFUSION AS A MANIFESTATION OF TUBERCULOSIS*

A Report of Seven Cases

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WALTHAM, MASSACHUSETTS

THE syndrome of alternating pleurisy with effusion is characterized by the development of serofibrinous pleurisy, at first unilateral and later contralateral. The onset and course of each effusion associated with this syndrome differ in no way from that of the more frequent purely unilateral type of pleurisy. Occurring as it does in the absence of pulmonary infiltration or with only a suspicion of it, this condition presents a diagnostic, therapeutic and prognostic challenge.

This syndrome has received scant clinical attention, and only isolated case reports have appeared. Gray[1] describes alternating pleurisy with effusion in an eighteen-year-old boy, with death resulting from miliary tuberculosis and tuberculous meningitis. Hurrell and Dawson-Walker[2] report a single case of alternating pleurisy with effusion occurring in a twenty-six-year-old man. Autopsy revealed generalized miliary tuberculosis. Gordon[3] briefly cites a case seen by Theodos of alternating pleural effusion with subsequent ascites developing in the presence of Pott's disease of the spine.

Hitherto, alternating pleurisy with effusion has been termed "bilateral pleurisy with effusion." It seems desirable to reserve the latter term for the rare cases of acute simultaneous bilateral pleurisy with effusion, thus demarcating the definite syndrome of alternating pleurisy with effusion.

Wilson[4] has summarized 14 cases of bilateral pleurisy with effusion. Of these, only 3 developed in the absence of demonstrable pulmonary infiltration and thus seemingly fall into the category under discussion, but it is not clear whether these cases represent alternating pleurisy with effusion or acute simultaneous bilateral pleurisy with effusion. It is probable that most cases of acute bilateral pleurisy with effusion actually represent alternating pleurisy with effusion, first discovered when bilateral effusion was present.

Cases of alternating pleurisy with effusion with definite evidence of widespread pulmonary tuberculosis are not included in the syndrome of alternating pleurisy with effusion, because the diagnosis is usually obvious. Effusions occurring during the course of bilateral therapeutic pneumothorax likewise are not considered, nor are the occasional cases of contralateral effusion developing after a unilateral artificial pneumothorax, with an accompanying effusion.

Acute serofibrinous pleurisy is of tuberculous origin in many, if not all, cases. Numerous follow-up studies, recently summarized by Farber,[5] attest to the serious ultimate prognosis of so-called serofibrinous pleurisy, an apparently benign and transient disease. Thirty to fifty per cent of cases of pleurisy with effusion later develop pulmonary tuberculosis. Occasionally miliary tuberculosis follows or accompanies such an effusion, and extrapulmonary tuberculosis is not infrequently a complication.

That alternating pleural effusion may be a manifestation of tuberculosis is shown by the following cases seen at the Middlesex County Sanatorium.

CASE REPORTS (FIGS. 1 and 2)

CASE 1. H. S. (M.C.S. 2155), a 30-year-old, married housewife, a sextigravida, when in the 5th month of pregnancy developed left-sided pleurisy and was hospitalized elsewhere on March 11, 1939, for 7 days. A roentgenogram of the chest showed a marked left-sided pleural effusion. A diagnostic thoracentesis was performed, and the fluid obtained was reported as positive for tuberculosis by guinea-pig inoculation. Because of recurrence of left-sided chest pain, the patient was readmitted elsewhere on March 29. A roentgenogram again showed a marked left-sided effusion. Following thoracentesis and further symptomatic treatment, she was discharged on April 12, somewhat improved.

The patient was first seen at the Diagnostic Clinic of the Middlesex County Sanatorium on April 24. A chest roentgenogram still showed a left-sided pleural effusion with cardiac displacement. Because she was in the 6th month of pregnancy, bed rest at home was advised. On July 30, acute right-sided pleuritic pain developed and she was again hospitalized elsewhere. A right-sided pleural effusion was present. On August 1, she delivered a normal baby, who died 1 week later.

The patient was admitted to this hospital on August 14 with a slight cough, minimal expectoration and a temperature of 103°F. A roentgenogram of the chest showed a right-sided pleural effusion, with slight haziness only at the left costophrenic angle. A right-sided thoracentesis performed on August 18 produced hazy amber fluid, which was positive for tuberculosis by guinea-pig inoculation. There was progressive improvement, and a roentgenogram taken on No-

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vember 15 showed only a small amount of fluid at the right base. The patient was discharged on March 16, 1940, after 216 days of residence.

When last seen on April 2, 1945, she was essentially well. A roentgenogram of the chest showed increased markings at the right base. Both costophrenic angles appeared normal.

Comment. There was an acute onset of left-sided pleural effusion, with fluid positive for tuberculosis by guinea-pig inoculation, in the 5th month of pregnancy. This was followed 4½ months later by acute right-sided pleural effusion, with the fluid again positive.

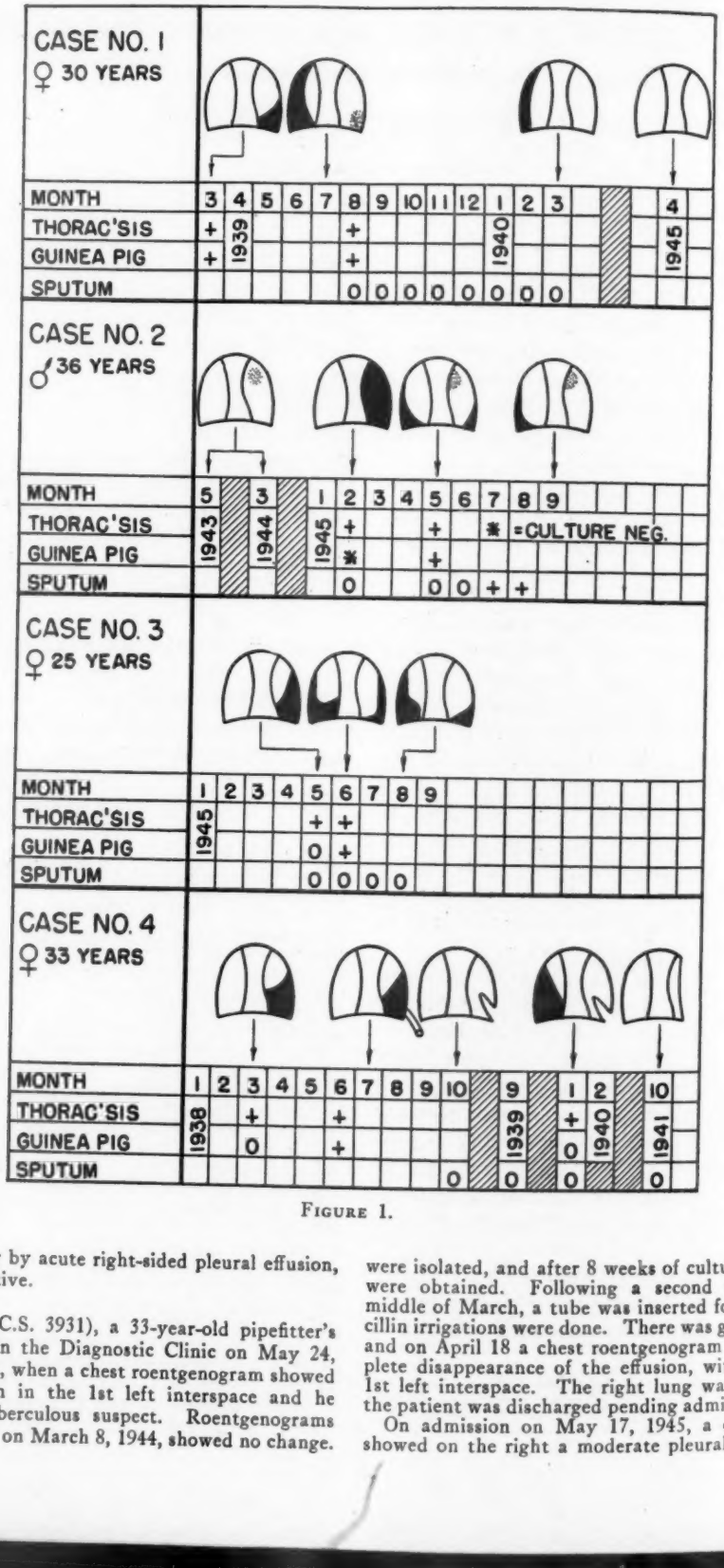
CASE NO. 1
♀ 30 YEARS
MONTH 3 4 5 6 7 8 9 10 11 12 1 2 3 4
THORAC'SIS + 1939 + 1940 1945
GUINEA PIG + + 1940 1945
SPUTUM 0 0 0 0 0 0 0 0
CASE NO. 2
♂ 36 YEARS
MONTH 5 3 1 2 3 4 5 6 7 8 9
1943 1944 1945
THORAC'SIS + + * =CULTURE NEG.
GUINEA PIG * +
SPUTUM 0 0 0 + +
CASE NO. 3
♀ 25 YEARS
MONTH 1 2 3 4 5 6 7 8 9
1945
THORAC'SIS + +
GUINEA PIG 0 +
SPUTUM 0 0 0 0
CASE NO. 4
♀ 33 YEARS
MONTH 1 2 3 4 5 6 7 8 9 10 9 1 2 10
1938 1939 1940 1941
THORAC'SIS + + + 0
GUINEA PIG 0 + 0
SPUTUM 0 0 0 0

FIGURE 1.

CASE 2. C. F. (M.C.S. 3931), a 33-year-old pipefitter's helper, was first seen in the Diagnostic Clinic on May 24, 1943, as a draft rejectee, when a chest roentgenogram showed questionable infiltration in the 1st left interspace and he was classified as a tuberculous suspect. Roentgenograms taken on August 25 and on March 8, 1944, showed no change.

The patient remained essentially well until February 9, 1945, when he developed acute malaise, chills and fever and rapidly became disoriented. A diagnosis of bronchopneumonia with left-sided pleural effusion was made by his physician after a chest roentgenogram, and he was admitted elsewhere. The sputum was negative for tubercle bacilli. Oral sulfadiazine and later intramuscular penicillin in adequate dosage were given, without apparent benefit. A left-sided thoracentesis was first done on February 28. No organisms were isolated, and after 8 weeks of culture no tubercle bacilli were obtained. Following a second thoracentesis in the middle of March, a tube was inserted for drainage and penicillin irrigations were done. There was gradual improvement, and on April 18 a chest roentgenogram showed almost complete disappearance of the effusion, with infiltration in the 1st left interspace. The right lung was clear. On May 5, the patient was discharged pending admission to this hospital.

On admission on May 17, 1945, a chest roentgenogram showed on the right a moderate pleural effusion and on the

left definite minimal infiltration in the 1st interspace, with thickened pleura at the base. No symptoms were associated with the appearance of this effusion. A right-sided thoracentesis performed on May 22 yielded 300 cc. of blood-tinged fluid, which was positive for tuberculosis by guinea-pig inoculation. On May 29, the fasting gastric content was negative for tuberculosis by guinea-pig inoculation, but routine 72-hour sputum concentrates on July 5 and August 14 were both positive for tubercle bacilli (Gaffky II) on a Ziehl–Neelsen smear. The patient is still hospitalized.

Comment. There was an acute onset of left-sided effusion, with a negative culture, followed by asymptomatic right-sided effusion, with the fluid positive by guinea-pig inoculation, in the presence of definite minimal left-sided infiltration that was previously only suspected. The interval between effusions was 3 months. The sputum eventually became positive.

Case 3. D. W. (M.C.S. 3927), a 25-year-old, unmarried Chinese woman developed a cough in February, 1945, which was diagnosed as bronchitis. On March 2, there developed slight pain in the thorax and acute diffuse abdominal swelling. The patient was admitted to this hospital on May 16, with moderate ascites and a small left-sided pleural effusion without parenchymal disease. A tuberculin test (0.01 mg.) was positive. The past history revealed that on May 12, 1943, because of vague abdominal complaints, laparotomy had been performed elsewhere. Considerable induration of the lower ileum was found. An appendectomy was performed, with removal of a cyst of the left ovary and of the left fallopian tube. Tuberculous peritonitis was considered but not proved. The pathological diagnoses were endometrial cyst of the ovary, chronic perioophoritis and questionable chronic appendicitis.

Clear, straw-colored fluid obtained by a left-sided thoracentesis on May 22 was negative for tuberculosis by guinea-pig inoculation. The aspirated gastric content on May 24 was negative for tuberculosis on guinea-pig inoculation. Specimens of the sputum were negative on concentration.

In early June, while the patient was still hospitalized, a slight cough, right-sided chest pain, dyspnea and a temperature of 100°F. developed. On June 9 a chest roentgenogram showed a massive right-sided pleural effusion, with considerable absorption of fluid on the left. Four hundred cubic centimeters of thin, slightly cloudy, straw-colored fluid obtained by thoracentesis on June 12 was positive for tuberculosis by guinea-pig inoculation. By June 19 there was no evidence of ascites. Despite further aspiration in an attempt to prevent fixation, a right-sided fibrothorax has developed, with marked limitation of right hemithoracic movement. The patient is still hospitalized.

Comment. The insidious onset of left-sided pleural effusion, with the fluid negative by guinea-pig inoculation, was coincidental with transient ascites, followed by the gradual onset of right-sided effusion, with the fluid positive. The interval between effusions was 3 months. Previous laparotomy suggested tuberculous peritonitis. There was eventual development of right-sided fibrothorax.

Case 4. M. O. (M.C.S. 2275), a 33-year-old, married housewife, developed a slight cough with pain in the left posterior chest in January, 1938, when 1 month pregnant. In March, malaise increased, slight fever began, and a heavy feeling developed in the left side of the chest. A roentgenogram taken on March 30 showed a left-sided pleural effusion. A left-sided thoracentesis performed elsewhere on March 31 yielded 480 cc. of fluid, which was negative for tuberculosis by guinea-pig inoculation. On May 29, vaginal bleeding began, and on June 2, incomplete miscarriage occurred. Curettage was done elsewhere on June 4, and convalescence was normal until June 17, when the temperature rose to 103°F. and a septic course developed. On June 25, a left-sided diagnostic thoracentesis yielded fluid that was positive for tuberculosis by guinea-pig inoculation. On July 6, the patient was transferred to another hospital, where a left-sided thoracentesis yielded fluid containing anaerobic nonhemolytic streptococci. On July 9, rib resection for tube drainage was done. A pleural biopsy showed tuberculous granulation tissue.

The patient was transferred to this hospital on July 29 with a diagnosis of mixed tuberculous and nonhemolytic streptococcus empyema, with no evidence of parenchymal disease, but left against advice on the 13th day.

Irrigation of the draining empyema cavity was done at home until September, when the temperature rose to 104° F. and the patient was hospitalized elsewhere. A secondary hemolytic streptococcus infection of the empyema cavity was found. The cavity had decreased in volume from about 200 cc. to 120 cc.

The patient was readmitted to this hospital on October 6. Empyema fluid was positive for tuberculosis by guinea-pig inoculation and also showed diphtheroids, streptococci and staphylococci. Frequent irrigations of the empyema cavity resulted in slow improvement. On February 14, 1939, a left-sided Schede thoracoplasty, with partial removal of the 6th, 7th, 8th, 9th and 10th ribs was done, but the incision broke down postoperatively. No further interference was advised because of the patient's condition, and she was discharged on September 21 after 351 days of residence.

Wound irrigations were continued at home. The patient remained fairly well until January, 1940, when she developed pain in the right side of the chest, cough, a temperature of 101°F., dyspnea and nausea. She was readmitted to this hospital on January 18 with a moderate right-sided pleural effusion. The left-sided thoracic incision was still open and draining. A right-sided thoracentesis on January 19 yielded 900 cc. of thin, cloudy, amber fluid, with the specific gravity 1.025, the red-cell count 637, and the white-cell count 360, with 1 per cent polymorphonuclear leukocytes, 96 per cent lymphocytes, 2 per cent eosinophils and 1 per cent undetermined forms. The fluid was negative for tuberculosis by guinea-pig inoculation. By April 9 there was only slight blunting of the right costophrenic angle with fluid. A three-stage left anterolateral thoracoplasty was begun on April 10 and completed on May 15. The patient did well postoperatively, the new incision healed fully, and closure of the old draining wound began. The sputum was never positive. She was discharged on October 1, 1941, after 623 days of residence.

By April, 1944, the old wound was fully healed without drainage. When last seen on April 9, 1945 the patient was essentially well but on limited activity. A roentgenogram of the chest showed a left-sided thoracoplasty with excellent lateral compression. The right lung was clear, with a normal-appearing right costophrenic angle.

Comment. There was an insidious onset of left-sided pleural effusion, with the fluid positive by guinea-pig inoculation, which after an incomplete miscarriage became secondarily infected and eventually required a left-sided Schede thoracoplasty. This was not successful and was followed by acute right-sided effusion, with the fluid negative by guinea-pig inoculation. The interval between effusions was 24 months. A left-sided thoracoplasty was required for final control. This catastrophic series of events could have undoubtedly been prevented by the present-day use of penicillin.[6]

Case 5. A. Z. (M.C.S. 2001), a 35-year-old, married housewife, with no known contact with tuberculosis, experienced the insidious onset of right-sided pleurisy in December, 1938. On January 8, 1939, she developed shaking chills and a temperature of 103°F. A roentgenogram of the chest taken elsewhere showed a right-sided pleural effusion from the 7th rib downward. A smear of the fluid showed no acid-fast organisms; 98 per cent of the cells were lymphocytes, and 2 per cent were polymorphonuclear leukocytes. Guinea-pig inoculation was not done. On February 15, when the patient was first seen in the Diagnostic Clinic, an attempted thoracentesis was unsuccessful.

The patient was admitted to this hospital on February 27, 1939. A 72-hour specimen of sputum taken on March 6, 1939, by the Greenfield–Anderson technic was positive for tubercle bacilli (Gaffky II), and stereoscopic roentgenograms on March 29 showed definite infiltration on the left, extending out from the upper pole of the hilum posteriorly to the 3rd rib and consistent with minimal pulmonary tuberculosis. This gradually retrogressed. Further sputum specimens remained negative. The patient was discharged on September 22 after 208 days of routine sanatorium care.

She remained well until November 19, when she developed acute left-sided chest pain, chills, a temperature of 102°F., cough and expectoration. Examination showed dullness to percussion with poor to absent breath sounds at the left base anteriorly and posteriorly. A chest roentgenogram taken on November 29 showed haziness at the extreme costophrenic angle on the right and a left-sided pleural effusion.

The patient refused readmission but took bed rest at home for 4 months. Thoracentesis was not done. By May, 1940, she was asymptomatic, and a roentgenogram showed only a small amount of fluid at the left base. In July, 1945, she was still well, and a roentgenogram showed the lungs to be clear and the costophrenic angles normal.

Comment. There was an acute onset of right-sided effusion and later transient left-sided infiltration, with a single sputum specimen positive. This was followed by acute left-sided effusion. The interval between effusions was 10 months.

CASE 6. M.T.B. (M.C.S. 3502), a 36-year-old, divorced woman, was first seen in the Diagnostic Clinic on November 23, 1939, because of contact with her brother, who was under treatment for pulmonary tuberculosis. A chest roentgenogram was negative. Because of fatigue and weight loss, roentgenograms were taken elsewhere in September, 1941, and a diagnosis of tuberculosis was made.

On October 2, 1941, the patient was admitted to this hospital. A tuberculin test (0.01 mg.) was positive. A roentgenogram showed mixed infiltration on the left, extending from the hilum to the 2nd interspace. Seven 72-hour sputum concentrates, two gastric aspirations with guinea-pig inoculation and a sputum inoculated into a guinea pig were negative. The patient left the hospital against advice on December 26 after 86 days of residence. After discharge she was seen periodically in the clinic and remained free of respiratory complaints, but in November, 1942, a diagnosis of pernicious anemia (later confirmed) was made elsewhere and injections of liver extract were begun. In October, 1943, she developed an unproductive cough and a temperature of 102°F., without chest pain. A massive left-sided pleural effusion with cardiac displacement was shown on examination and by chest roentgenogram.

The patient was readmitted on November 3, 1943. A left-sided thoracentesis yielded 600 cc. of Burgundy-red fluid, which was negative by guinea-pig inoculation. Fluid aspirated on November 12 and 27 was also negative. Sputum concentrates were consistently negative. Liver injections were begun, and the red-cell count rose from 3,430,000, with a hemoglobin of 78 per cent (Sahli), on admission to 4,000,000 on April 4, 1944, and to 4,850,000, with a hemoglobin of 88 per cent, on discharge. On March 7, the fasting gastric content was negative by guinea-pig inoculation. On routine sanatorium regimen the patient progressed satisfactorily until April 18, when severe cough, right-sided chest pain, shortness of breath and a temperature of 102°F. developed coincidentally with a right-sided pleural effusion. A roentgenogram showed persistent effusion on the left and moderate effusion on the right. On April 24, 600 cc. of amber cloudy fluid was aspirated from the right side of the chest. On May 1 and 15, a right-sided thoracentesis yielded 850 cc. and 200 cc., respectively, of clear, yellow fluid. No tubercle bacilli were demonstrated on smear. Guinea pigs were not available, but on each occasion the fluid was negative for tubercle bacilli on culture. The temperature became normal within 8 weeks, and there was slow improvement. The patient was discharged at her own request on November 3 after 367 days of residence. A chest roentgenogram taken at that time showed thickened pleura or fluid at both bases, extending upward toward the axillas, with mixed infiltration

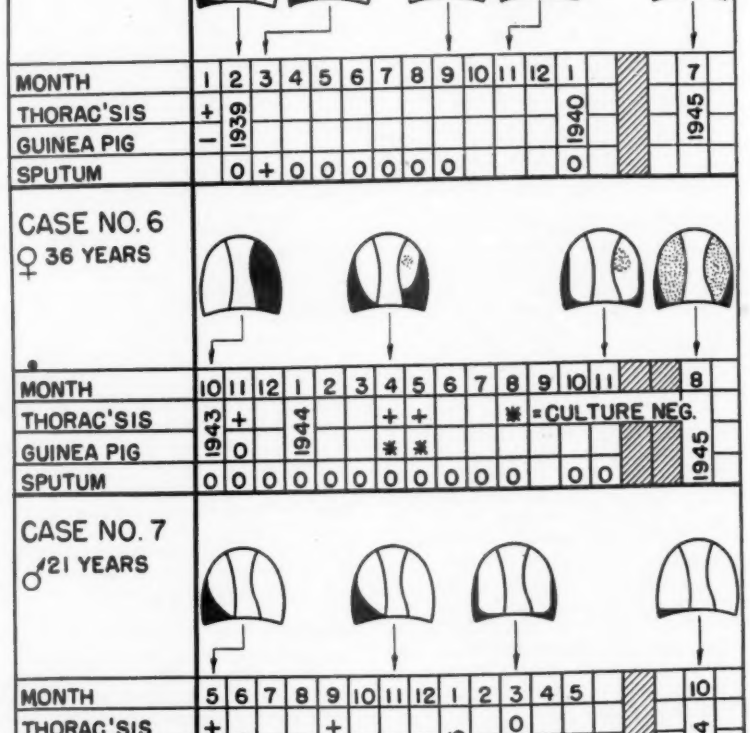

FIGURE 2.

about the left hilum extending into the 2nd and 3rd interspaces.

The patient failed to keep regular clinic appointments, and when seen on August 27, 1945, she had developed severe cough, expectoration, ascites and edema of the ankles. A chest roentgenogram showed exudative infiltration throughout both lung fields, with persistence of marginal pleural reaction consistent with miliary tuberculosis. Sanatorium readmission was advised, but the patient died at home on September 1. Permission for autopsy was not obtained.

Comment. There was an acute onset of left-sided effusion, with the fluid negative by guinea-pig inoculation, in the presence of suspicious left-sided hilar infiltration, with repeated negative sputum and gastric contents. This was followed by acute right-sided effusion, with a negative culture. The interval between effusions was 6 months. There was eventual development of miliary pulmonary lesions. The cause of death was not ascertained but was undoubtedly tuberculous.

Case 7. O. P. (M.C.S. 796), a 21-year-old, married Negro cook, experienced the gradual onset of a slightly productive cough, dyspnea on exertion, a temperature in April, 1934, of 103°F. and weight loss. He was hospitalized elsewhere on May 9, when a chest roentgenogram showed a massive right-sided pleural effusion with cardiac displacement. Thoracentesis on May 11, 1934, yielded yellow-red fluid, with the specific gravity 1.020, the red-cell count 7,000 and the white-cell count 0. By May 28 the fluid had become yellow, with the specific gravity 1.018, the red-cell count 4,000 and the white-cell count 500, all lymphocytes. Intradermal injection of old tuberculin (0.1 mg.) was positive. Guinea-pig inoculation of aspirated fluid was negative for tuberculosis. Ten sputum examinations showed no acid-fast organisms.

On August 31, the patient was transferred to this hospital. A chest roentgenogram showed a right-sided pleural effusion, with no evidence of parenchymal disease. A right-sided thoracentesis performed on September 8 yielded 50 cc. of clear, greenish fluid, which was negative for tubercle bacilli by guinea-pig inoculation and on culture using Parker's medium. The patient was discharged on November 23 after a total hospital residence of 218 days, 85 of which were spent in this hospital.

He continued well until March 18, 1935, when a small asymptomatic effusion blunting the left base appeared. A thoracentesis performed on April 18, 1935, was unsuccessful. The patient was closely followed in the clinic, and by May 28 the fluid had disappeared. He continued asymptomatic and on October 10, 1944, was essentially well. A chest roentgenogram taken at that time showed a horizontal right diaphragm, but the lungs were clear.

Comment. There was a gradual onset of right-sided effusion, with the fluid negative by guinea-pig inoculation. This was followed by an asymptomatic transient left-sided effusion. Thoracentesis was unsuccessful. The interval between effusions was 10 months.

Discussion

The apparent incidence of alternating pleurisy with effusion is low. Among 3822 patients with pulmonary tuberculosis admitted to this sanatorium between September 31, 1931, and September 1, 1945, only 7 cases of alternating pleurisy with effusion were found. During this same interval 48 patients with pleurisy with effusion were admitted. Since Amberson[7] has estimated that in 5 per cent of all cases of pulmonary tuberculosis the serous membranes are involved, it is obvious that only a small percentage of patients with serofibrinous pleurisy are admitted for sanatorium treatment. This is presumably due to the general failure to appreciate both the serious prognosis of serofibrinous pleurisy and the need for sanatorium treatment. Hence, although the statistical incidence of alternating pleurisy with effusion is low, the syndrome is undoubtedly more frequent than is generally realized.

The pathogenesis of alternating pleurisy with effusion is presumably hematogenous. Tuberculous bacillemia is more frequent than is usually appreciated.[8]

Diagnosis

In the syndrome described, the onset and course of the pleurisy with alternating effusion differed in no way from the onset and course of unilateral serofibrinous pleurisy.[5]

Serofibrinous pleurisy may be of acute or insidious onset, with malaise, fever, cough, variable expectoration and often dyspnea. Ordinarily the active phase of the effusion lasts for four to six weeks, sometimes longer. Occasionally the effusion is transient and asymptomatic, being discovered during routine examination.

The cases presented above show features characteristic of those accepted as clinically diagnostic of so-called serofibrinous pleurisy.[7] As with unilateral pleurisy with effusion, extrapulmonary tuberculosis may precede, accompany or follow alternating pleurisy with effusion.

Thoracentesis is indicated to establish the nature and etiology of a pleural effusion in the absence of obvious systemic disease. The fluid of serofibrinous pleurisy is an exudate. The cells are predominantly lymphocytes, but rarely the fluid is hemorrhagic.[9] Importance, but not pathognomonic significance, can be attached to such a lymphocytosis, for effusions due to carcinoma and to pulmonary emboli may also show this feature.[9]

Guinea-pig inoculation or culture of aspirated fluid is definitely indicated in serofibrinous pleurisy with effusion. Examination of the smear alone for tubercle bacilli is insufficient. In a series of 119 consecutive cases of pleural effusion, occurring chiefly during the course of therapeutic pneumothorax, from different cases routinely studied at the Middlesex County Sanatorium in 1941–1942 and found to be positive for tuberculosis, the fluid in 45 cases was positive — usually Gaffky II or more — on smear (Ziehl–Neelsen stain), and guinea-pig inoculation was accordingly not done. In 7 cases, the fluid was positive on smear and also positive by guinea-pig inoculation. In 67 cases, it was negative on smear but positive by guinea-pig inoculation. Furthermore, of the 24 patients admitted to this sanatorium from 1931 to 1940, inclusive, with a diagnosis of pleurisy with effusion, thoracentesis yielded fluid that was positive (Gaffky II) on smear (Ziehl–Neelsen stain) in only 1 case. In 10 cases the fluid was negative on smear but positive for tuberculosis by guinea-pig inoculation. In 7 cases, it was negative both on smear and by guinea-pig inoculation. In 6 cases, the result of examination was not reported.

Amberson[10] has pointed out that concentration of a larger volume of pleural fluid before guinea-pig

inoculation can logically be expected to increase the percentage of positive tests. On the other hand a negative result of guinea-pig inoculation does not exclude a tuberculous origin. A later aspiration may yield fluid that is positive. In Case 4, the fluid originally aspirated from the left side of the chest was negative for tuberculosis by guinea-pig inoculation but was positive three and a half weeks later. Similarly, in the case reported by Hurrell and Dawson–Walker[2] both fluids originally aspirated were negative but later were positive. Furthermore, as shown in Table 1, there is a high incidence of

TABLE 1. *Results of Guinea-Pig Inoculation of Pleural Fluid in Pleurisy with Effusion.*

AUTHOR	NO. OF CASES	POSITIVE*	NEGATIVE*
Bonilla[11]	22	7 (2)	15 (7)
Gaarde[12]	79	22 (15)	57 (26)
Totals	101	29 (17)	72 (33)
Percentage of cases developing tuberculosis		58	45

*The figures in parentheses represent patients who later developed tuberculosis, chiefly pulmonary.

pulmonary tuberculosis following the development of serofibrinous pleurisy, despite negative results of guinea-pig inoculation.

A final diagnostic procedure that should be used more frequently than at present is guinea-pig inoculation of the fasting gastric content or of sputum if it is being produced. Infrequent cases with a positive guinea-pig inoculation of the gastric content or of sputum in the presence of negative pleural-fluid findings, with no roentgen-ray evidence of pulmonary infiltration, have been observed at this sanatorium.

Differential Diagnosis

There are numerous causes of pleural effusion. Tinney and Olsen[13] have analyzed 274 cases seen at the Mayo Clinic, with the results indicated in Table 2.

Alternating pleurisy with effusion occurring in the course of such diseases as congestive failure,

TABLE 2. *Proved Etiology of Pleural Effusion.*

ETIOLOGY	NO. OF CASES	PERCENTAGE
Carcinoma	141	51
Congestive failure	42	15
Lymphoma	28	10
Pneumonia	24	9
Tuberculosis	16	6
Cirrhosis of liver	8	3
Chronic nephritis, with nephrosis	7	3
Miscellaneous conditions	8	3
Total	274	

pulmonary infection, polyserositis (Concato's disease), metastatic carcinoma, lymphoma, cirrhosis, septicemia, rheumatic fever or nephritis should be easily distinguished from the syndrome described above and thus offer no difficulty.

A nontuberculous bacterial etiology of the syndrome under discussion has been excluded, since routine cultures of the effusions were negative except, of course, in Case 4, in which was a secondary infection.

Certain relatively infrequent diseases may have to be considered on occasion.

That alternating pleural effusion may be an incident in the course of a polyserositis associated with disseminated lupus erythematosus is admitted. Not one of the cases summarized above, however, showed suggestive skin lesions, leukopenia, albuminuria, pericardial effusion, joint symptoms or other evidence of the characteristic widespread polyserositis associated with disseminated lupus. Furthermore, after variable follow-up periods no suggestive symptoms have developed.

Pulmonary emboli with accompanying effusion may conceivably give rise to the syndrome of alternating pleural effusion. Careful physical examination, with special reference to calf tenderness, presence of Homans's sign and alteration in the diameter of the calf, together with consideration of the associated and characteristic roentgenographic findings,[14] should easily exclude such a possibility.

Severe nutritional disturbances with hypoproteinemia or marked vitamin B deficiency may give rise to serous effusions that are rarely pleural and may possibly simulate the syndrome under discussion.

Chronic constrictive pericarditis — frequently of tuberculous origin — with the characteristic symptoms of dyspnea, abdominal enlargement and often ankle edema may be accompanied by pleural effusion. The fluid is usually a transudate. Similarly, since the fluid of Meigs's[15] syndrome is characteristically a transudate, no diagnostic confusion should arise even though the course of this disease may mimic alternating pleural effusion. Finally, Loeffler's syndrome, characterized by transient, recurrent pulmonary infiltration, a parasitic infestation or an allergic background, a benign clinical course and eosinophilia, has recently been shown to be a rare cause of bilateral pleural effusion and hence a potential cause of alternating pleurisy with effusion.[16] Thus, a twenty-one-year-old man with characteristic infiltration in both lungs developed bilateral pleural effusion. Culture and guinea-pig inoculation were both negative for tubercle bacilli. The fluid from the right side contained 29 per cent eosinophils, and that from the left side contained 64 per cent eosinophils, which appears to be a distinguishing feature.

Exclusion of any of the above conditions should not be difficult if one is familiar with the nature of serofibrinous pleurisy with effusion.

Treatment

As in acute serofibrinous pleurisy with effusion, the early treatment is symptomatic. Thoracentesis

is indicated initially for diagnosis and to visualize the underlying lung parenchyma, to relieve respiratory, cardiac or gastric embarrassment produced mechanically and occasionally to help in hastening fluid resorption.

Although the acute phase of serofibrinous pleurisy ordinarily lasts for four to six weeks, several months of a sanatorium regime is an essential part of proper treatment. Amberson[7] states that serofibrinous pleurisy with effusion should be treated as is active tuberculosis, with sanatorium care. Trudeau[17] has shown that the prognosis of pleurisy with effusion with either negative or minimal apical pulmonary findings is excellent in patients receiving at least four months of sanatorium treatment. Gaarde's[12] findings are in agreement. Obviously, alternating pleurisy with effusion, a variety of acute serofibrinous pleurisy and a manifestation of tuberculosis, should be treated in a comparable fashion, with prolonged bed rest.

Chest roentgenograms taken at least semiannually for a period of five years are part of proper follow-up supervision if possible parenchymal infiltration is to be discovered early. Since the early infiltration of pulmonary tuberculosis is notoriously silent, both symptomatically and clinically, it is only by frequent roentgenograms that pulmonary tuberculosis following serofibrinous pleurisy can be discovered before extensive progression has taken place.

Prognosis

Alternating pleurisy with effusion may be a disease of serious ultimate significance in the absence of proper treatment. Although no conclusions can be drawn from this small group of cases, it appears that a sanatorium regimen or its equivalent is necessary if the likelihood of eventual development of pulmonary or extrapulmonary tuberculosis is to be reduced to a minimum. Death from miliary tuberculosis may be expected in a certain portion of cases of alternating pleurisy with effusion, irrespective of sanatorium treatment.[1, 2]

Summary and Conclusions

Seven cases of alternating pleurisy with effusion, defined as the development of serofibrinous pleurisy occurring in the absence of pulmonary infiltration or only with suspicion of it and followed after a variable period of time by the development of contralateral serofibrinous pleurisy, are presented.

The onset and course of each effusion associated with this syndrome differ in no way from that of the more frequent unilateral serofibrinous pleurisy.

It is shown that alternating pleurisy with effusion may be a manifestation of tuberculosis.

The proper treatment of pleurisy with effusion includes a prolonged sanatorium regimen.

References

1. Gray, J. W. Bilateral effusion followed by miliary tuberculosis of lungs and tuberculous meningitis. *Lancet* **1**:75, 1940.
2. Hurrell, G., and Dawson-Walker, E. F. Bilateral tuberculous effusion in miliary tuberculosis of lungs. *Lancet* **2**:313, 1939.
3. Gordon, B. Incidence and mechanism of bilateral pleural effusions. *J. A. M. A.* **114**:733-735, 1940.
4. Wilson, G. C. Bilateral tuberculous pleurisy with effusion: analysis of 14 cases. *Am. Rev. Tuberc.* **39**:745-747, 1939.
5. Farber, J. E. Prognosis in cases of serofibrinous pleurisy. *New Eng. J. Med.* **228**:784-787, 1943.
6. Bird, K. T., Bushueff, B. P., and Dawson, F. P. Pyopneumothorax: treatment of two cases with penicillin. *Am. Rev. Tuberc.* **53**:122-128,1946.
7. Amberson, J. B., Jr. Tuberculosis of the serous membranes. A chapter in Cecil, R. L. *A Textbook of Medicine.* Sixth edition. 156 pp. Philadelphia and London: W. B. Saunders Company, 1943. P. 302.
8. Wilson, G. S. *Tuberculosis Bacillemia.* Med. Res. Coun. Spec. Rep. Series No. 182, London, 1933. 146 pp.
9. Berliner, K. Hemorrhagic pleural effusion: analysis of 120 cases. *Ann. Int. Med.* **14**:2266-2284, 1941.
10. Amberson, J. B., Jr. *1938 Year Book of General Medicine.* 840 pp. Chicago: Year Book Publishers, Inc. P. 286.
11. Bonilla, J. L., Jr. Pleurisy with effusion. *Am. Rev. Tuberc.* **46**:59-66, 1942.
12. Gaarde, F. W. Prognosis of pleurisy with effusion. *J. A. M. A.* **95**: 249, 1930.
13. Tinney, W. S., and Olsen, A. M. Significance of fluid in pleural space: study of 274 cases. *J. Thoracic Surg.* **14**:248-252, 1945.
14. Hampton, A. O., and Castleman, B. Correlation of postmortem chest teleoroentgenograms with autopsy findings with special reference to pulmonary embolism and infarction. *Am. J. Roentgenol.* **43**:305-326, 1940.
15. Meigs, J. V., and Cass, J. W. Fibroma of ovary with ascites and hydrothorax, with report of seven cases. *Am. J. Obst. & Gynec.* **3**:249-267, 1937.
16. Baumann, H. Eosinophile Pleuritis bei fluchtigem, eosinophilen Lungeninfiltrat. *Schweiz. med. Wchschr.* **74**:326, 1944.
17. Trudeau, F. B. Pleural effusion: its prognosis in patients showing little or no parenchymal involvement. *Am. Rev. Tuberc.* **39**:57-63, 1939.

INTESTINAL OBSTRUCTION FOLLOWING ABDOMINAL BATTLE WOUNDS

LIEUTENANT COLONEL PHILIP S. FOISIE, M.C., A.U.S.*

IN A previous paper,† the management of colostomies secondary to battle wounds of the abdomen has been discussed. Although most of these patients have done remarkably well during their definitive treatment at this general hospital, a few have shown complications that sometimes follow the damage caused by the passage of a ragged shell fragment through the abdomen. These cases should serve as a warning signal to the physicians under whose care such patients may later come.

The necessity of operating on a few of these soldiers has made us conscious of the potential danger of intestinal obstruction. We have been amazed at times that these patients have been able to maintain a functioning intestinal stream in spite of the amount of constriction and angulation of bowel that was found. In such patients particularly, early signs and symptoms suggesting the possible onset of obstruction must receive careful study and evaluation.

Anyone who reads the often penciled and sometimes blood-stained notes of the Field Medical Records is at once struck with the fact that some of these patients lived to reach a general hospital. These records frequently describe intestinal trauma of a severity foreboding almost certain death. The credit for such survivals belongs to the prompt and skillful surgical procedures of the forward medical installations, to the giving of plasma and blood by persons in this country and to the near-miracles of chemotherapy.

Factors that predispose to the formation of adhesions are multiple bowel perforations and lacerations of the bowel, particularly when these have resulted in considerable peritoneal soiling, hemorrhage, blast, lacerations of the mesentery, delay prior to initial surgery and infection. Since battle injuries may involve all these factors, it is not surprising that subsequent obstruction often results.

When some of these patients have been operated on for acute obstruction, an interesting phenomenon in the mechanism of the obstruction has been noted. In civil practice, intestinal obstruction is usually caused by some single discrete difficulty — a strangulated hernia, volvulus, intussusception or the like. The obstruction in these patients, however, is likely to occur partially at several points, with a gradual damming-up effect that increases to the final point of complete obstruction. This building-up of back pressure progresses in the reverse peristaltic direction toward the stomach, until at last so much distention occurs that the bowel becomes twisted or sharply angulated at a point of fixation and total obstruction results. The mechanism of obstruction is therefore generally that of a series of obstructive points, no one of which is severe enough to cause total obstruction but that together produce a cumulative back pressure, with increasing stagnation of the intestinal stream, until complete obstruction ensues. With the usual obstruction from a single source, correction of the offending mechanism at the point where the bowel changes from distention to collapse solves the problem. In cases with the mechanism described above, on the other hand, the relief of the area of total obstruction frequently only leads one on to another point a few centimeters away distally at which highly questionable function is again encountered.

There is a constant danger in cases of this sort that an enthusiastic surgeon will do too much. The natural desire to free the bowel completely should be curbed, attention being confined to the few points at which some degree of definite obstruction has occurred. Inflamed, fibrin-covered bowel that has been freed is likely to form new adhesions, which may prove a greater hazard than those already freed. A totally obstructed patient should be subjected to as little trauma as will accomplish the goal of the operation — namely, re-establishment of the intestinal stream. Once the obstruction has been relieved, functioning bowel, even though densely adherent, should be left alone. More extensive lysis of adhesions may be attempted later, under more favorable conditions, if symptoms warrant.

Of the many substances that have been used to prevent reformation of adhesions, amniotic fluid has probably been the most successful. It was not available at this hospital, but may be of value in the subsequent treatment of these and similar cases in which further operative procedures are indicated. In several cases, the patients' condition halted mobilization of the bowel before it appeared certain that we had done all that was necessary, but their recovery justified this decision. In other words, a "let well enough alone" policy is applicable to these cases. In some of them we have been surprised on exploration to find with what severe matting together of the entire bowel patients had been able to maintain function until the acute obstructive episode occurred. In some cases, the bowel was so severely involved that complete freeing at a single operative procedure would have been impossible, but once the acute obstructive points were relieved these patients did well. They are, of course, liable to develop similar complications in the future, as are many others who have had intra-abdominal trauma but have so far been able to avoid difficulty. It is safe to predict that in veterans who have suffered abdominal wounds, acute intestinal obstruction will be encountered for many years to come.

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†Foisie, P. S. Closure of colostomies. *New Eng. J. Med.* 234:464-468, 1946.

Five case histories will be cited to illustrate the surgical problem involved.

Case 1. A soldier was wounded in August, 1944, by high-explosive shell fragments, which blew out a large segment of the sacrum, tearing the rectum and causing multiple lacerations of the small bowel. He was operated on at an evacuation hospital through a long midline incision, and several defects in the ileum were repaired. Colostomy was not done at that time. He arrived at this hospital 2 weeks after his injury, at which time most of the feces were being evacuated through the sacral wound. The day after admission a Mikulicz colostomy was performed to divert the fecal stream from the wound. This effort was successful, and with irrigations the wound became clean and was completely healed in 8 weeks, during which time the patient's general condition improved.

The colostomy spur was then crushed and the colostomy was closed. Normal bowel movements followed these procedures, and the patient was apparently ready for evacuation when he developed a sudden and complete obstruction. This event was accompanied by such profound shock that several hours of resuscitative measures were required to prepare the patient for surgery. Under local anesthesia, the colostomy was reopened to ensure complete drainage of the lower bowel. This did not give relief, and after further supportive treatment the abdomen was reopened through a midline incision. Practically the entire bowel was found to be tangled in a mass of adhesions, with only the jejunum and the transverse and descending colon free. With a proximal approach, as much of the small bowel was freed as the patient's condition permitted. This soon proved to be futile, since several portions of the bowel were gangrenous. A catheter was sutured into the most distal point of free, viable small bowel, and the abdomen was closed. The prognosis was obviously hopeless.

At autopsy, performed 2 days later, the entire small bowel from the catheter onward was twisted in a gangrenous mass, which was firmly adherent to the pelvis and flank and also involved the ascending colon. The whole mass had to be resected to disentangle it completely.

Comment. This case, the only one in the present series that terminated fatally, demonstrated the extreme involvement of bowel that may occur.

Case 2. The patient, a 20-year-old soldier, was wounded on November 12, 1944, by mortar-shell fragments, which caused multiple injuries, including penetration of the abdomen and multiple wounds of the small bowel. There was considerable inter-abdominal bleeding. At operation on November 22 at an evacuation hospital, the lacerated bowel was sutured. Colostomy was not done. Infection of the abdominal wound ensued.

On admission to this hospital on December 9, the patient was running a septic type of temperature and complained of pain in the right inguinal region. Chemotherapy and supportive treatment were used, pending a clearer indication for surgery, until December 13, when acute localized tenderness developed in the right lower quadrant of the abdomen, with definite muscle spasm. Laparotomy was performed on this date through the old wound, which was paper thin and was not completely healed. A diffuse fibrinous peritonitis was found, with free pus throughout the abdomen, multiple small walled-off abscesses and severe matting of the bowel by extensive adhesions. The abscess cavities were broken up, and drains were inserted through a stab wound into the pelvis. Although obstruction seemed unavoidable, the patient never developed any signs of it and became afebrile within 10 days. He was evacuated to the Zone of Interior on January 6, 1945, in good general condition.

Comment. This case, in which there was no actual obstruction, illustrates well the peritoneal difficulties that may follow abdominal battle wounds. There was every reason to expect obstruction, which demonstrates what extensive disease may be tolerated, with maintenance of intestinal flow, provided that the adhesions are innocuous. This patient is of course liable to have subsequent complications.

Case 3. A 23-year-old soldier was wounded in Germany on December 7, 1944, by high-explosive shell fragments, which caused multiple wounds, including an abdominothoracic injury with severe right hemothorax, perforation of the right diaphragm and liver and considerable intra-abdominal bleeding, but no bowel perforation. He was operated on at a field hospital on the day of the injury. The abdominal wound was closed after the evacuation of considerable blood, and drainage to the right subphrenic area was established through a stab wound in the flank. Thoracotomy was done, with repair of the rent in the right diaphragm.

The patient was admitted to this hospital on December 25 and appeared to be convalescing satisfactorily. On December 29, he developed sudden severe pain in the left lower quadrant of the abdomen and shortly presented signs of total intestinal obstruction. Laparotomy revealed multiple adhesions, which tied the bowel at several places, from the ileocecal valve through the distal third of the ileum to a point at which a loop of small bowel was anchored and twisted into a volvulus in the left lower quadrant. A loop of terminal ileum was firmly adherent at a point deep in the pelvis, and the freeing of this loop completely relieved the obstruction. The patient made an excellent recovery, in spite of being given doughnuts and coffee by a fellow patient on the 3rd postoperative day.

Comment. This case demonstrates the development of a total obstruction secondary to abdominal injury with no bowel perforation. The adhesions apparently developed from a retained blood clot.

Case 4. The patient, a Negro soldier, sustained abdominal wounds, which had been treated at another general hospital for several weeks. On the way to the Zone of Interior, while lodged for the night at this hospital, he developed abdominal pain, distention, vomiting and acute localized tenderness and spasm. Obstruction was considered certain. Laparotomy disclosed the multiple-gradient type of obstruction described above, with the final point of total obstruction at a point in the left flank, to which a loop of twisted small bowel was securely fixed by a dense adhesion. Section of these adhesions gave complete relief.

Case 5. A 28-year-old soldier was wounded on April 18, 1945, by a rifle bullet, which entered the right buttock and transversed the abdomen, making its exit through the left lower quadrant of the anterior wall. In its course the lower end of the right ureter was severed 3 cm. above the vesical orifice. Two loops of small bowel were sufficiently injured to require resection and end-to-end anastomosis. There were also a compound fracture of the right ilium and extensive retroperitoneal hemorrhage.

The initial bowel repair was done at an evacuation hospital on the day of injury. The patient was subsequently treated at another general hospital, and was about to be evacuated to the Zone of Interior when the station hospital was ordered closed and he was transferred here to this hospital.

At admission on May 22, the patient presented a soft abdomen, with a nearly healed abdominal scar showing some evidence of recent infection. There was a urinary fistula through the right buttock.

One week after admission, colicky abdominal pain, with nausea, vomiting and increasing distention suddenly developed. Wangensteen suction gave no relief, and since the symptoms were rapidly progressing, operation was performed 3 hours after the onset of the acute attack. The old midline scar was excised, and when the abdomen was opened a generalized fibrinous peritonitis was found. This was most acute in the right lower quadrant, where multiple loops of bowel were matted together, apparently in an attempt to wall off urinary leakage from the severed ureter. The bowel, including the large bowel as far down as the sigmoid, was distended.

The adherent loops in the right lower quadrant were freed, a catheter was sutured into the cecum and brought out through a stab wound, and the wound was closed with heavy silk sutures without drainage. Ochsner treatment was instituted, with 40,000 units of penicillin in each infusion.

On the 10th postoperative day, the patient was doing well. The abdomen was soft, with no distention. Flatus was being expelled by rectum, and the temperature, pulse and respirations were normal. It was planned, as soon as the patient's condition permitted, to drain the kidney pelvis so as to remove the source of the peritonitis.

Comment. Although much of the distention was due to peritonitis, it seems probable that this patient had an acute obstruction due to the fact that the distended bowel was

caught in adhesions in the right lower quadrant, secondary to urinary leakage.

Summary

Abdominal battle injuries are likely to produce extensive adhesions that pave the way for subsequent obstruction. Such patients may do well for varying periods, but a serious hazard persists, and obstructive episodes will probably be encountered for years to come. Early signs and symptoms should receive careful evaluation and close watching.

A graduated obstructive mechanism is described. With acute obstruction only the offending mechanism should be freed. Interval lysis with the instillation of amniotic fluid may be advisable when chronic symptoms suggest impending difficulty. Five illustrative cases of acute intestinal obstruction, with 1 death, are reported.

SUCCESSFUL TREATMENT OF SEVERE DIABETIC COMA COMPLICATED BY CHRONIC LARYNGEAL OBSTRUCTION*

Report of a Case

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THE association of severe diabetic coma and chronic laryngeal obstruction is rare. The case reported herein presented these two problems, and in addition a third, a severe mental depression following tracheotomy that induced the patient to neglect the treatment of his diabetes. Although his immediate survival depended on recovery from the coma, his return to life as a useful citizen awaited the repair of the tracheotomy. In the treatment of the coma, the largest amount of insulin ever reported to have been given in twenty hours was administered. Three delicate plastic operations were performed to repair the tracheotomy. Our experience with such a patient is worth recounting, in the hope that it may be a means of rescuing others from death or despondency.

There are many reports in the medical literature of extreme hyperglycemia requiring tremendous doses of insulin. These cases can be divided into two groups. In the first, hyperglycemia is usually associated with coma and its cause is evident — infection, hepatic disease, endocrine disorder or neglect of insulin therapy or diet. The acidosis is controlled by large doses of insulin, and the patient usually returns to his precoma status. In the second group, in which also the condition may be associated with coma, there is no explanation for the obstinate hyperglycemia, and the need for excessive doses of insulin continues for weeks or even months. This group is composed of patients who may be called insulin-resistant. It will not be further discussed here.

The case reported presents several unusual features. The coma was precipitated by infection and neglect of insulin therapy. The infection was apparently a severe, nonspecific gastroenteritis. The patient received 3620 units of insulin in twenty hours. In Wiener's[1] case the patient required 3250 units of insulin in the emergency, but the case differed from the present one in his continued need for large doses of insulin. In a case reported by Glass, Spingarn and Pollack,[2] the patient required 85,000 units over a five-and-a-half-month period and the maximum twenty-four-hour dose was 2795 units. It is interesting that the excessive insulin need subsided six months after its onset. The greatest daily quantity of insulin given at the New England Deaconess Hospital has been 2272 units in the case of a man who had diabetic coma during a phase of insulin resistance.[3] Many cases, however, have been reported in which the initial blood-sugar levels exceeded that of 1240 mg. per 100 cc. in the present case. The highest value recorded is 2060 mg.[4] Dillon and Dyer[5] reported the highest blood-sugar level (1850 mg.) recorded in a patient who survived.

Case Report

C. D., a 34-year-old man, a native of Massachusetts, was admitted to the Massachusetts General Hospital for the fifth time on January 3, 1945, semicomatose and in severe diabetic acidosis.

The patient was first admitted to this hospital on April 10, 1929, for regulation of his diabetes, which had been discovered in 1924, when he was 14 years old. In 1925, his physician had started him on insulin, gradually increasing the dose and varying the diet to suit the patient's needs. He did not co-operate well and was therefore hospitalized. After 10 days of study, he was discharged on a diet consisting of 120 gm. of carbohydrate, 180 gm. of protein and 100 gm. of fat and a dosage of 50 units of insulin (20-20-10).

He did well thereafter until 1935, when he was readmitted to the Orthopedic Service with a fracture of the right tibia and fibula and phenobarbital poisoning. At this time the diabetes was slightly more difficult to control, and the patient was discharged on a dosage of 51 units of crystalline insulin (25-5-15-6) and a diet consisting of 160 gm. of carbohydrate, 92 gm. of protein and 101 gm. of fat.

The third admission occurred in 1936, and was for tonsillectomy and diabetic regulation on protamine-zinc insulin. There had been some abnormal neurologic findings, — sluggish knee and ankle jerks and equivocal plantar responses, — and lumbar puncture revealed an elevated spinal-

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fluid protein. Accordingly, the patient was thought to have an associated diabetic neuritis. His course had been essentially uneventful, however, and he was discharged on a dosage of 40 units of protamine-zinc insulin and a diet consisting of 129 gm. of carbohydrate, 69 gm. of protein and 76 gm. of fat.

The patient was regularly followed in the Diabetes Clinic for the next 3 years. He then disappeared for 3 years, reappearing in the Emergency Ward in April, 1943, with severe diabetic acidosis, precipitated by a throat infection. The blood sugar was 434 mg. per 100 cc., the carbon dioxide content 18.5 millimols per liter, and the blood nonprotein nitrogen 64 mg. per 100 cc. He was given 388 units of crystalline insulin (124 units intravenously), 4500 cc. of 5 per cent dextrose in normal saline solution and 15 gm. of sodium bicarbonate. The acidosis responded well, but the patient ran a stormy course and developed severe respiratory distress, which necessitated an emergency tracheotomy on April 28, 1943. He was later found by x-ray to have complete laryngeal obstruction. Throat cultures showed a hemolytic *Staphylococcus aureus* to be predominating, and he was therefore given sulfadiazine for 2 weeks. Since the tracheotomy incision could not be closed because of the laryngeal obstruction, he was discharged on June 22 with the tube in place. The insulin requirement at that time was 30 units of protamine-zinc insulin and 10 units of crystalline insulin every morning, with occasional extra crystalline insulin by test.

In the following November, the patient was readmitted for widening of the tracheotomy. Three operations were later performed in an unsuccessful attempt to close the tracheotomy permanently. At that time it was extremely difficult to regulate the diabetes. The patient was discharged on a dosage of 40 units of globin insulin and a diet of 225 gm. of carbohydrates, 85 gm. of protein and 90 gm. of fat.

Throughout the next year, he appeared at irregular intervals in the Diabetes Clinic and was seen there on August 4, at which time the urine showed an orange color on a Benedict test.

The patient's living conditions had been extremely poor. He lived alone and prepared his own meals, living chiefly on canned foods and paying little attention to the prescribed diet. He was unwilling to work at his former trade of weaving because the tracheotomy made him self-conscious, and his sole interest in life was to return to the hospital to have it repaired. As months went by without word of an appointment, he became more and more depressed and lost the desire to live. He denied alcoholic indulgence during this time, but admitted almost complete neglect of his diabetes treatment.

The fifth admission occurred on January 3, 1945, when the patient was brought to the Emergency Ward by ambulance in a semicomatose condition. Two days previously he had developed abdominal pain, nausea, vomiting and diarrhea. He had been taking insulin — 80 units of globin insulin — "fairly regularly" but had not tested his urine for some weeks. The diet had consisted largely of canned foods in irregular amounts. The night before admission he called a physician, who suspected an impending diabetic coma but gave no extra insulin. On the morning of admission, in addition to the symptoms listed, the patient had suddenly become deaf and was unable to hear unless one shouted directly into either ear.

The physical examination revealed a thin, pale and semicomatose patient. The skin and tongue were dry. The eyeballs were soft. The breath smelled of acetone. The patient could be aroused only with difficulty and spoke in a hoarse voice when he closed the tracheotomy tube. There were a few coarse rales at both lung bases, but no abnormal breath sounds. The heart was not enlarged. The rhythm was totally irregular, and the heart sounds were of poor quality. No murmurs were heard. The abdomen was soft, but there was generalized tenderness without rebound or spasm. The liver, spleen and kidneys were not felt. Deep-tendon reflexes were absent on admission.

The temperature was 95.8°F., the pulse 140, the respirations 40 (Kussmaul), and the blood pressure 80/50.

The urine gave a brick-red test for sugar and ++++ tests for acetone and diacetic acid and contained a slight trace of albumin. Its specific gravity was 1.022. The hemoglobin was 12.7 gm. per 100 cc., the red-cell count 4,300,000, and the white-cell count 22,700. The blood-sugar level was 1240 mg. per 100 cc. and the carbon dioxide was 3.8 millimols per liter. The nonprotein nitrogen was 108 mg. per 100 cc., the chlorides 76 milliequiv. per liter, and the total protein 8.2 gm. per 100 cc.

Therapy was started immediately with 1500 cc. of normal saline solution intravenously, 100 units of crystalline insulin — 50 units intravenously and 50 subcutaneously — and 15 gm. of sodium bicarbonate. The stomach was aspirated, but no fluid was obtained. This was a rather hazardous procedure, and the patient struggled against it so much that he became cyanotic owing to improper functioning of the tracheotomy tube. An indwelling catheter was inserted shortly after admission, and 1000 cc. of urine was obtained. A second infusion was started with 250 cc. of blood plasma in saline. When the blood chemical findings became available 1½ hours after admission the insulin dosage was increased to 100 or 200 units intravenously and 100 or 200 units subcutaneously hourly; thus during the first 3 hours the patient received a total of 720 units. Despite the giving of constant intravenous saline solution with alkali, he remained anuric for 6 hours. Accordingly, blood chemical analyses were checked at hourly intervals. After an initial fall from 1240 mg. per 100 cc. to 928 mg., the blood-sugar level remained at approximately 900 mg. for 4 hours, necessitating constantly increasing doses of insulin. At 2:30 p.m., 4 hours after admission, the patient became extremely cyanotic and dyspneic. The pulse could not be obtained, and the respirations ceased. Oxygen and artificial respiration were given, and 3 cc. of a 25 per cent solution of Coramine and 0.5 gm. of caffeine sodium benzoate were injected intravenously, after which the respirations and pulse returned. The insulin dosage was increased to 200 units every half hour — 100 units intravenously and 100 subcutaneously — in a desperate attempt to lower the blood sugar and correct the ketosis. At 4:30 p.m. an increase in the number of rales in the lungs was noted, and in view of the auricular fibrillation, which was confirmed by electrocardiogram, digitalization was begun at once with 6 cc. (1.2 mg.) of Cedilanid intravenously. Shortly after this, 5 cc. of urine was obtained. In an attempt to produce a diuresis, 20 cc. of a 10 per cent solution of serum was given at 5:10 p.m. Thereafter the patient's condition steadily improved, the insulin dosage was lowered, and at 4 a.m., 18 hours after admission, the blood sugar was 119 mg. per 100 cc., the carbon dioxide 37.1 millimols per liter, the nonprotein nitrogen 72 mg. per 100 cc., and the chloride 95 milliequiv. per liter. The last dose of insulin (50 units) was given at 3 a.m. At 6:30 a.m. there was a mild insulin reaction, with the blood sugar 35 mg. per 100 cc., which was relieved by 50 cc. of 50 per cent glucose intravenously. The summary of therapy during the first 20 hours is given in Table 1. The

TABLE 1. *Summary of Therapy Given During the First Twenty Hours.*

SUBSTANCE	QUANTITY
Crystalline insulin	3620 units (⅓ intravenously, ⅔ subcutaneously)
Normal saline solution	7000 cc. (intravenously)
Sodium bicarbonate	52.5 gm. (intravenously)
Blood plasma	1500 cc. (intravenously)
Whole blood	250 cc. (intravenously)
Cedilanid (Lanatoside C)	1.6 mg. (intravenously)
Coramine	0.75 gm. (intravenously)
Caffeine sodium benzoate	0.5 gm. (intravenously)
10% saline solution	20 cc. (intravenously)
Broth and fruit juice	800 cc. (orally)

total fluid intake during the first 20 hours was 9630 cc., and the output was 2035 cc., exclusive of the 1000 cc. obtained when the patient was catheterized shortly after entry.

On the following day, the patient continued to complain of abdominal pain and was unable to eat. He was fed by intravenous fluids and required only 40 units of insulin throughout the day. The hearing had returned to normal. The heart rhythm had also returned to normal, — this was confirmed by electrocardiogram, — and it was thought that digitalis was no longer necessary. Blood-sugar samples taken throughout the day varied between 52 and 171 mg. per 100 cc., and the carbon dioxide had begun to return to normal (Table 2).

The abdominal pain persisted for 4 days, but on January 7 the patient was able to eat and an attempt to regulate the diabetes was begun. This proved to be quite difficult, and various combinations of protamine-zinc, crystalline and

globin insulin were tried. The disease was finally under fairly good control with 35 units of protamine-zinc insulin every morning. On this dosage the urine varied between green, with sediment, and blue, the fasting blood sugar was 105 mg. per 100 cc. on one occasion and 152 mg. on another.

On February 16, 45 days after admission, the patient was transferred to the Massachusetts Eye and Ear Infirmary for a plastic operation on the trachea, in an attempt to close the tracheotomy and thus remove the factor responsible for the mental depression. Two operations were performed in the preparation of skin flaps on the right and left sides of the neck. In the right-hand flap several strips of cartilage were embedded to give added support to the trachea. The tube was left in place, and on April 7 the patient was discharged hospital. Deafness was an unusual complication of the coma and could not be explained. No reference to this concomitant appears in the literature. Another unusual feature was the occurrence of auricular fibrillation, which so far as we know has been reported in only 1 other case of diabetic coma.[3] In both cases the fibrillation was transient and recovery from the coma was complete.

It has been suggested, on review of the data, that this patient was overtreated. This is admitted, but with qualification. The insulin dosage was large but not excessive. If larger doses had been given early the total dose required might have been smaller. Doubtless larger doses should have been administered at once, even before the blood analysis was reported. The dosage of sodium bicarbonate was perhaps excessive, but it caused no irreversible changes. The fluid intake, in view of his severe dehydration and nausea, was not excessive. The question of whether the blood and plasma were necessary is one of pure speculation. The patient's condition was such that they could not well have been omitted. Likewise, the oxygen, Coramine, caffeine, Cedilanid and hypertonic saline solution were given under definite indications. Each of

TABLE 2. *Insulin Dosage and Blood Chemical Findings.*

DATE	TIME	DOSE OF INSULIN	BLOOD CHEMICAL FINDINGS				
			SUGAR	CARBON DIOXIDE	NONPROTEIN NITROGEN	CHLORIDE	TOTAL PROTEIN
		units	*mg./100 cc.*	*mM./l.*	*mg./100 cc.*	*mE./l.*	*gm./100 cc.*
1/3/45	10:30 a.m.	50	1240	3.8	108	76	8.2
	11:00	50					
	12:00 noon	120	928	5.2			
	12:30 p.m.	100					
	1:00	200	920	7.9	108		
	1:30	200					
	2:00	100	900	6.0			
	2:30	100					
	3:00	200	920	9.9			
	3:30	200					
	4:00	200	776	10.6		82	6.2
	4:30	200					
	5:00	200	652	12.8	93		
	5:30	200					
	6:00	100	652	16.3			
	6:30	100					
	7:00	100	592	19.3	79	88	
	7:30	100					
	8:00	100	532	21.4			
	8:30	100					
	9:00	100	436	24.2	80	93	
	9:30	100					
	10:00	100	376	26.7			
	10:30	100					
	11:00	100	326	29.3	71	91	6.2
	11:30	50					
	12:00 midnight	50					
1/4/45	12:30 a.m.	50					
	1:00	50	220	33.9			
	1:30	50					
	2:00	50					
	2:30	50					
	3:00	50					
	4:00		119	37.1	72	95	
	6:30		35	36.7			
	8:30		131	38.2	47	101	5.5
	12:45 p.m.		103	36.7	46	100	
	4:30		52	37	40		
	9:00		171	34.3			
1/5/45	9:00 a.m.		109	34.8	37	102	5.5
	3:00 p.m.		200	30.9			
1/6/45	9:00 a.m.		33	32.4	30	92	5.7
	5:00 p.m.		260	24.5			

home on a dosage of 30 units of protamine-zinc insulin and 15 units of globin insulin every morning and a diet of 300 gm. of carbohydrates, 100 gm. of protein and 80 gm. of fat.

On June 2, the patient was readmitted to the Eye and Ear Infirmary, where the tracheotomy was successfully closed at a third operation by incising around the edges of the opening and suturing them over it, and swinging the previously prepared delayed flap from the right side of the neck over the tracheotomy.* No change was made in the satisfactory program used for the regulation of the diabetes, and on June 20 the patient was discharged asymptomatic and in good spirits.

DISCUSSION

This patient had a severer diabetic acidosis and required more insulin in the first twenty-four hours than has any other patient ever treated in this

*These operations will be described in detail in a later publication by Dr. V. H. Kazanjian, to whom we are indebted for this account.

these probably contributed to the patient's recovery from a moribund state, as did also the artificial respiration.

The reconstruction of the air passages, — the trachea and larynx, — an extremely delicate but successful therapeutic procedure was of great importance in the maintenance of morale and of the desire for life after recovery. At first it was suggested that the patient's condition presented too great a risk or at least promised too short a survival time to warrant the hazard, but the reverse seemed likelier to be true — namely, that the repair of the air passages would make life again worth living, and hence that so far as the diabetes, and with it the patient's longevity, was concerned, the risk of not operating would actually be greater than the risk of operating. This prophecy has so far proved to be correct.

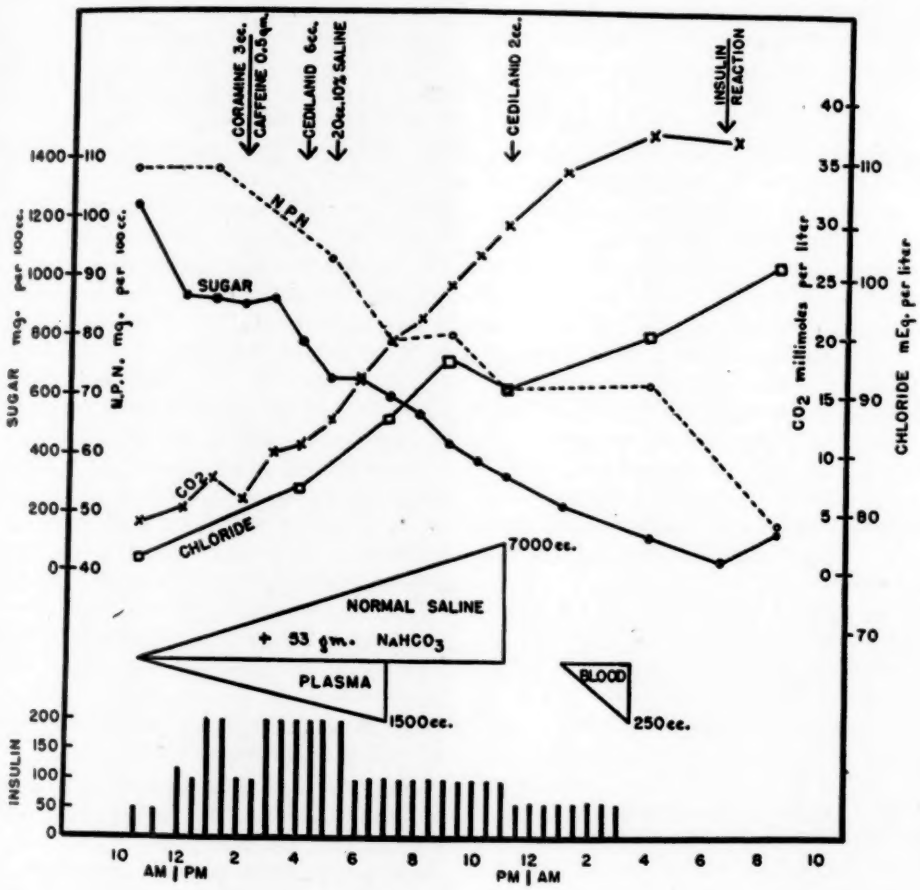

FIGURE 1.

repair the air passages were carried out to rehabilitate him after recovery from diabetic coma. This has undoubtedly been as important in aiding the patient's longevity as was the immediate treatment of the diabetic coma.

SUMMARY

The case is reported of a man of thirty-four in diabetic coma who required for recovery 3620 units of insulin in twenty hours. Other unusual features were deafness and auricular fibrillation, both of which quickly cleared up.

Because the patient was prone to neglect his daily treatment as a result of a mental depression caused or aggravated by a long persisting tracheotomy, a series of three delicate plastic operations to

REFERENCES

1. Wiener, H. J. Diabetic coma requiring unprecedented amount of insulin: report of case manifesting extreme insulin resistance. *Am. J. M. Sc.* **196**:211-217, 1938.
2. Glass, W. I., Spingarn, C. L., and Pollack, H. Unusually high insulin requirements in diabetes mellitus: report of case. *Arch. Int. Med.* **70**:221-235, 1942.
3. Bailey, C. C. Treatment of severe diabetic coma with 1,820 units of insulin. *J. A. M. A.* **121**:255, 1943.
4. Lawrence, R. D. Extreme hyperglycaemia in diabetic coma. *Brit. M. J.* **1**:377, 1934.
5. Dillon, E. S., and Dyer, W. W. Diabetic coma with extreme hyperglycemia. *Am. J. M. Sc.* **190**:683-686, 1935.

MEDICAL PROGRESS

SURGERY OF THE HEART AND STRUCTURES RELATED TO IT

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SURGERY of the heart and structures related to it will be considered under five main headings — congenital lesions, trauma, acute pericarditis, constrictive pericarditis and miscellaneous conditions.

CONGENITAL LESIONS

Pulmonary Stenosis or Pulmonary Atresia

Blalock and Taussig[1] have recently reported and discussed 3 cases in which children with severe degrees of pulmonary stenosis were operated on successfully and with apparent benefit. Heretofore, there has been no satisfactory treatment for pulmonary stenosis or pulmonary atresia. A so-called "blue baby" with a malformed heart was considered beyond the reach of surgical aid. These authors in undertaking the operation were convinced that even though the structure of the heart was grossly abnormal, it might be possible in many cases to alter the course of the circulation in such a manner as to lessen the cyanosis and the resultant disability. They emphasize the fact that it is not the cyanosis per se that does harm.

In the development of this thesis, the studies of Lundsgaard and Van Slyke[2] on the causes of cyanosis are cited. These studies demonstrated the great significance of pulmonary factors. The extent of the oxygenation of the blood in the lungs clearly depends on the vital capacity of the patient, the rate of flow of blood through the lungs, the partial pressure of the oxygen in the inspired air and a specific pulmonary factor that is designated as the "alpha factor." In most, if not all, cases in which there was a pronounced polycythemia, there were secondary changes in the lungs of such a nature that all the blood that passed through them was no longer in effective contact with the oxygen in the alveoli. The fact that all the blood that circulated through the lungs was not fully oxygenated made it seem improbable that if more blood had circulated through the lungs a larger proportion of it would have been oxygenated. Blalock and Taussig believe that this demonstration of the alpha factor completely overshadows another vitally important factor — namely, the volume of blood that reaches the lungs for aeration.

Expressed in the simplest terms, the circulation of the blood through the lungs after birth is essential for life; anyone deprived of such circulation dies. For example, in all cases of pulmonary atresia in which the circulation to the lungs is by way of the ductus arteriosus, the closure of the latter renders the condition incompatible with life.

Blalock and Taussig then discuss a variety of congenital malformations that illustrate the importance of the volume of the pulmonary circulation in the production of cyanosis. One of these is the malformation consisting of a single ventricle with a rudimentary outlet chamber. If the great vessels occupy their normal positions, the aorta arises from the common ventricle and is of large caliber, whereas the pulmonary artery arises from the rudimentary outlet chamber and is of small caliber. As a result, a large volume of blood goes to the systemic circulation and only a small volume of blood goes to the lung, and there is intense cyanosis. If the great vessels are transposed and the pulmonary artery is large and the aorta is small, a large volume of blood goes to the lungs for aeration, and cyanosis is minimal or absent.

Another case in point is that of truncus arteriosus. When the pulmonary arteries arise directly from the aorta, there is adequate circulation to the lungs, and cyanosis is minimal or absent. If, however, the pulmonary artery fails to arise from the heart or to connect with the aorta and the circulation to the lungs is by way of the bronchial arteries, only a small volume of blood reaches the lungs for aeration, and cyanosis is intense.

As a result of these and other observations, these authors conclude that many gross malformations of the heart are compatible with life provided there is adequate circulation to the lungs and, furthermore, that lack of circulation to the lungs is the primary cause of death in many infants with congenital malformation of the heart. An appreciation of these facts, together with an extensive previous experience with the experimental use of large arteries for the purpose of conducting blood to sites not usually supplied by such vessels, led to the development of an operation for increasing the flow of blood through the lungs in patients with congenital malformations of the heart. The operation consists in making an anastomosis between a branch of the aorta and one of the pulmonary arteries, — in other words, the creation of an artificial ductus arteriosus.

Levy and Blalock[3] have demonstrated the feasibility of anastomosing a systemic artery to one of the pulmonary arteries in experimental animals. Blalock and Park[4] have reported the suturing of the severed proximal end of the subclavian artery to the aorta as a means of conducting blood beyond

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the point of an experimental coarctation of the aorta. Since the operation to be described was devised to compensate for an inadequate flow of blood to the lungs, it seemed desirable that the anastomosis be made in such a manner that the blood from the systemic artery would be enabled to reach both lungs. Suture anastomosis could not be made to the main pulmonary artery, since occlusion of this vessel for more than a few minutes causes death. It therefore appeared that the anastomosis should be made just distal to the division of the main pulmonary artery and, furthermore, that the side of the chosen vessel should be used so that the blood might flow to both lungs.

With such an extensive clinical and experimental background, briefly summarized above, Blalock operated successfully on 3 patients, all of them children, who had a clinical diagnosis of tetralogy of Fallot with a severe degree of pulmonary stenosis. There were slight variations in each of the operations, but the following abbreviated description summarizes the major features.

Under light general anesthesia, an anterior transpleural approach was made on the right or left side, depending on the position of the great vessels and the artery to be used in the anastomosis. The right or left pulmonary artery was exposed and dissected from the adjacent tissues for as great a distance as possible. The subclavian or innominate artery was dissected free of the adjacent tissues, and by the use of a bulldog arterial clamp the vessel chosen was occluded temporarily at the point at which it arose from the aorta. In the case of the innominate artery, its branches were ligated at their origins and it was cut across just proximal to the ligatures. A bulldog arterial clamp was placed on the right or left pulmonary artery just distal to the point of division of the main pulmonary artery. A second clamp was placed on the right or left pulmonary artery just proximal to the point where the vessel gave off a branch to the upper lobe of the lung. A transverse opening was made into the side of the pulmonary artery midway between the two clamps. The anastomosis between the end of the systemic artery and the side of the pulmonary artery was then carried out, with a meticulous technic and with the employment of fine silk on a curved needle as suture material. The bulldog clamps were removed from the pulmonary artery, and this was followed by removal of the clamp from the systemic vessel. The lung was re-expanded, and the incision in the chest wall was closed.

Each of the 3 patients suffered from such a severe degree of pulmonary stenosis that there was inadequate circulation to the lungs. Although the three operations differed in detail, in each case the procedure greatly increased the volume of blood that reached the lungs.

Fear that an intensely cyanotic patient would not tolerate a long operative procedure in which it was necessary to open the pleural cavity and to occlude temporarily one of the pulmonary arteries proved unfounded. Fortunately, the administration of oxygen during anesthesia increased the oxygen content of the arterial blood, and cyanosis was definitely lessened. The question whether a patient already severely anoxemic would tolerate the occlusion of one of the main pulmonary arteries for the period necessary in performing the anastomosis appears to have been answered satisfactorily. The 3 patients tolerated occlusion of the left or right main pulmonary artery for periods of thirty to ninety minutes.

Another question that arose was whether ligation and division of the left subclavian artery or the innominate artery would result in serious impairment of the circulation to the arm and brain. Blalock and Taussig were gratified to note that there was little evidence of impairment of circulation to the parts deprived of their major arterial pathway. The pulse was absent for some time postoperatively, and the part was slightly cooler than the corresponding one on the opposite side of the body, but immediately after operation it was evident that the circulation was adequate to maintain life in the part. The advisability of performing upper dorsal sympathectomy at the time of operation, which does not add to the burden of operation, is suggested by the authors.

Blalock and Taussig state that the degree of impairment in the flow of blood to the lungs varies from patient to patient and that the selection of the vessel to be used depends on the extent of the need of the patient for an increase in the circulation to the lungs. The size of the vessel chosen and of the anastomosis itself should not be larger than is necessary for the relief of anoxemia, because of the danger associated with excessive shunting of blood to the lungs.

A discussion of other methods by which an anastomosis between the systemic and pulmonary circulations may be made includes a simple and rapid method that Blalock and his associates[3, 4] have carried out in experimental animals. The first portions of the medial walls of the aorta and the pulmonary artery, which are intimately adherent to each other, are contained within the pericardial cavity and are enclosed in a tube of serous pericardium common to the two vessels. They were able to produce a fistula between the two vessels in dogs by inflicting a stab wound in this region. The knife blade was introduced through the opposite free wall of the pulmonary artery, the walls of the pulmonary artery and aorta, which were in intimate contact, were pierced, the knife was withdrawn, and the opening in the free side of the pulmonary artery was closed by sutures. The establishment of the fistula required only a few seconds. The method is mentioned because it may be necessary to use the major blood vessels and to employ considerable speed if

newborn infants with pulmonary stenosis or atresia are to be saved.

As to indications, Blalock and Taussig are of the opinion that the types of abnormalities that should be benefited by the operation described are the tetralogy of Fallot and pulmonary atresia, with or without dextroposition of the aorta and with or without defective development of the right ventricle. Other abnormalities likely to be benefited are a truncus arteriosus with bronchial arteries and a single ventricle with a rudimentary outlet chamber in which the pulmonary artery is diminutive in size. The operation is indicated only when there is clinical and radiologic evidence of a decrease in the pulmonary blood flow.

They believe that the operation is not indicated in cases of complete transposition of the great vessels or in the tetralogy of Fallot of so-called "Eisenmenger type," and probably not in aortic atresia. It should not be performed when studies reveal a prominent pulmonary conus or pulsations at the hili of the lungs.

Coarctation of the Aorta

Crafoord and Nylin[5] have recently reported 2 cases of coarctation of the aorta that were cured by surgical means. Apparently these operations, which were performed in October, 1944, are the first successful ones to be done for this lesion in man. Blalock and Park,[4] however, had already described a method of by-passing the coarctation, which they had carried out successfully in experimental animals.

Because Crafoord had demonstrated that the flow of blood to all the organs could remain suspended for as long as twenty to twenty-five minutes without subsequent signs of organic damage, provided there was an adequate flow of blood to the brain, he took the risk of placing clamps on the aorta above and below the patent ductus arteriosus and left them in place during the time necessary to divide the ductus and suture the aorta. In one patient the aorta was thus occluded just below the origin of the left subclavian artery for twenty-seven minutes without noticeable subsequent disturbances of organs. It was on the basis of this experience in patients with patent ductus arteriosus that Crafoord conceived his operation for coarctation of the aorta, and because of the relatively bad prognosis in these cases he felt justified in making an attempt at surgical treatment. Excerpts from Crafoord's description of his operation follow.

> In both patients, the aorta showed conical narrowing from the origin of the subclavian artery, and the conical constriction between the coarctation and the point of departure of the subclavian artery was much longer in the first case [a small boy] than in the second. . . . Clamp forceps were then attached to the aorta just below the point of origin of the subclavian artery, as well as at a point as far away as possible from the first pair of aortic intercostal arteries. Small clamp forceps were then placed on all the branches arising from the aorta between the two forceps on the main trunk of the aorta. Resection of the aorta, taking in the most constricted segment was then carried out. On the peripheral side, the incision through the aorta was made in a plane at right angles to the long axis of the aorta, 2 mm. proximal to the point of origin of the first pair of intercostal arteries. Centrally, the place of incision was placed at an angle of about 45° in relation to the long axis of the aorta, owing to the fact that the lumen here was narrower than on the peripheral aspect, and about 1 cm. away from the area of greatest constriction. In this way, two lumens with circumferences to fit one another were obtained. A typical Carrel aorta suture was then made. First, three interrupted sutures were placed at equal distances from each other, and then running silk sutures were sewn between the interrupted sutures, each stitch being drawn up separately to secure adequate tautness.
>
> When the forceps were removed, it was found that an ideal artery suture with no leaks had been achieved.

The patients were both in excellent health when examined in March, 1945.

Crafoord and Nylin consider it remarkable that in both cases normal conditions as regards the blood pressure were re-established. In both the blood pressure in the legs was a little higher than that in the arms, and no true hypertension was present. They believe that this postoperative effect is in favor of the mechanical theory of hypertension in coarctation.

Working independently, Gross and Hufnagel[6] evolved an almost identical technic on dogs, and operated on two patients in June and July, 1945. The first patient, a six-year-old child, died when the clamps were removed from the aorta, the heart going into uncontrollable dilatation as the result of the sudden opening of the enormous vascular bed. The second patient, a twelve-year-old girl, survived the operation and was discharged in a satisfactory condition on the nineteenth postoperative day. In this case the clamps were gradually released over a ten-minute period. In view of their experience in their first case, the authors stress the importance of the slow removal of the aortic clamps.

Alexander and Byron[7] report the successful removal of an aneurysm of the thoracic aorta. This is apparently the first time that an aneurysm of the thoracic or abdominal aorta has been successfully removed or that the thoracic aorta has been successfully ligated. The patient, a nineteen-year-old student, showed a nonpulsating mass in the left side of the mediastinum, mild hypertension and a well established collateral circulation. A provisional diagnosis of aortic coarctation on the basis of extrinsic pressure by a neoplasm — probably a neurofibroma — was made, and thoracotomy was performed. At operation a thin-walled saccular aneurysm of the upper descending aorta was exposed in the costovertebral gutter. Its upper pole was 5 cm. inferior to the left subclavian artery, and its attachment to the aorta was 7.5 cm. long. The aorta appeared to be slightly constricted at its junction with the upper end of the aneurysm. Since the thin-walled aneurysm was in danger of rupture and since the collateral circulation appeared to be adequate, resection of the aneurysm

was undertaken. The aorta was ligated with 1.3 cm.-wide cotton tape just below the left subclavian artery and 4 cm. below the aneurysm. The divided ends of the aorta were oversewed with silk.

The convalescence was uneventful until the seventeenth day, when acute cardiac decompensation occurred. This was treated satisfactorily with oxygen, morphine, venesection, digitalization, mercupurin and ammonium chloride. The patient was discharged on the thirty-sixth postoperative day.

The pathogenesis of the aneurysm was not definitely established, but the authors consider it necessary to assume the presence of a coarctation to explain the remarkably well developed collateral circulation, the bilateral rib notching and the absence of pulsation of the aneurysm and of the aorta below it.

The success of this operation was attendant on a happy combination of circumstances, and it is extremely unlikely that this combination will again be encountered. As the authors point out, this case was unique in that all the factors essential to the success of the operation were present — youth and general good health, the absence of syphilitic disease of the aorta, the presence of a well established collateral circulation and the favorable location of the aneurysm, which permitted the placing of the proximal ligature on the aorta below the left subclavian artery.

The authors also emphasize that the success in their case has no bearing on the management of aneurysm in general.

Patent Ductus Arteriosus

The literature on patent ductus arteriosus continues to increase, and as might be expected with the availability of larger series of cases that have been treated by ligation, certain authors have attempted to determine the need for surgery in these cases. Shapiro and Keys[8] discuss a clinical series of 51 cases and analyze operations undertaken for ligation of the patent ductus in 140 cases. During their years of observation, they have been led to consider the lesion an apparently harmless one. Children have gone through school, young men have done hard labor and women have borne children, all with no evidence of cardiac disability. On the basis of these observations, one may readily assume an attitude of conservatism. Analysis of the world literature, however, showed that 80 per cent of patients eventually die from their cardiac lesion. Those who were alive at seventeen years of age averaged thirty-five years at death. At least 40 per cent of these patients died of subacute bacterial endarteritis, and most of the remainder died of congestive heart failure. The average age of the authors' series of 51 patients was less than twenty-five years, and the oldest patient died at fifty-eight. These data force reconsideration of a conservative attitude toward what might seem radical surgery. The authors conclude that surgery is advisable for most patients with patent ductus arteriosus, with or without subacute bacterial endarteritis. Ligation should be attempted immediately once a diagnosis of subacute bacterial endarteritis has been made.

Burch[9] states that ligation should be done if there is associated endarteritis, and also if there is progressive cardiac damage and decompensation or definite impairment of mental and physical development. He reviewed most of the reported cases of surgical ligation and found the mean mortality rate to be 8.5 per cent in uninfected cases. Burch believes that in the future the operation will probably be recommended as soon as a patent ductus is discovered, but in view of the mortality no such recommendation can be made now. Poor results following ligation, he states, may be attributed to failure to find the ductus, ligation of the wrong vessel, wound sepsis, hemorrhage, associated cardiovascular abnormalities and incomplete obliteration of the ductus.

Such a reactionary attitude does not seem fully justified. The operation is still technically in its developmental stage, and most thoracic surgeons are of the opinion that a considerable reduction in the operative mortality is by no means beyond achievement. Of course, only time will tell how ligation will affect the life span.

The effect of ligation on infection of a patent ductus has been dramatic, and infection must now be considered an urgent indication for operation. Tubbs[10] describes 9 cases of subacute bacterial endarteritis complicating this anomaly and treated by ligation. Six of the patients were well fifteen months to four years after operation. He stresses the importance of the early diagnosis of the supervention of infection so that ligation may be undertaken at the optimum time, before the infection has had an opportunity to render the ductus friable and the adjacent tissues edematous.

Bettman and Tannenbaum[11] report the successful ligation of a patent ductus complicated by bacterial endocarditis in a woman eighteen years old. She made a good recovery and was well two years after operation. Harper and Robinson[12] treated a similar case in a woman of twenty-eight who had long-standing severe subacute bacterial endocarditis. The ductus was ligated with two silk ligatures, and cellophane was wrapped loosely about it. These authors believe that the cellophane was responsible for the final complete occlusion of the ductus. The murmur and symptoms disappeared for two weeks, but reappeared and persisted for two and a half months after operation. The murmur and symptoms then completely disappeared, and the patient remained entirely well for one and a half years after surgery. The final, complete and permanent occlusion of the patent ductus arteriosus was thought to have been brought

about by the cellophane. The application of cellophane is not a new maneuver, but its use under similar circumstances is worth while for the possible continuing stimulation of fibrosis within the ductus.

(To be concluded)

References

1. Blalock, A., and Taussig, H. B.: Surgical treatment of malformations of heart, in which there is pulmonary stenosis or pulmonary atresia. *J. A. M. A.* **128**:189-202, 1945.
2. Lundsgaard, C., and Van Slyke, D. D. *Cyanosis.* 80 pp. Baltimore: Williams & Wilkins Company, 1923.
3. Levy, S. E., and Blalock, A. Experimental observations on effects of connecting by suture left main pulmonary artery to systemic circulation. *J. Thoracic Surg.* **8**:525-530, 1939.
4. Blalock, A., and Park, E. A. Surgical treatment of experimental coarctation (atresia) of aorta. *Ann. Surg.* **119**:445-456, 1944.
5. Crafoord, C., and Nylin, G. Congenital coarctation of aorta and its surgical treatment. *J. Thoracic Surg.* **14**:347-361, 1945.
6. Gross, R. E., and Hufnagel, C. A. Coarctation of aorta: experimental studies regarding its surgical correction. *New Eng. J. Med.* **233**: 287-293, 1945.
7. Alexander, J., and Byron, F. X. Aortectomy for thoracic aneurysm. *J. A. M. A.* **126**:1139-1145, 1944.
8. Shapiro, M. J., and Keys, A. Prognosis of untreated patent ductus arteriosus and results of surgical intervention. *Am. J. M. Sc.* **206**: 174-183, 1943.
9. Burch, G. E. Congenital patent ductus arteriosus: evaluation of its surgical treatment. *M. Clin. North America* **28**:388-402, 1944.
10. Tubbs, O. S. Effect of ligation on infection of patent ductus arteriosus. *Brit. J. Surg.* **32**:1-12, 1944.
11. Bettman, R. B., and Tannenbaum, W. Ligation of patent ductus arteriosus in presence of apparent bacterial endocarditis: report of case apparently cured. *Ann. Int. Med.* **21**:1035-1037, 1944.
12. Harper, F. R., and Robinson, M. E. Occlusion of infected patent ductus arteriosus with cellophane. *Am. J. Surg.* **64**:294-296, 1944.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

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CASE 32151

Presentation of Case

First admission. A sixty-five-year-old railroad conductor was referred from another hospital with a diagnosis of multiple abscesses of the lung.

Eight months before admission the patient had an appendectomy, with drainage because of rupture, at a community hospital. Two weeks later an abscess in the region of the cecum was drained. This was soon followed by an episode of chills and sweating, accompanied by a high fever and pain in the right lower chest. A small amount of pus was removed from the right pleural cavity. The patient received sulfonamides and penicillin and was discharged apparently well in the seventh week. Two weeks after discharge he again developed chills and fever and was readmitted. The empyema was treated by drainage and local penicillin, and he was discharged after a four-week stay. Six weeks later he entered another hospital, again with chills and fever. There was a draining sinus in the right lower posterior chest. Injection of the sinus with lipiodol showed a multilocular abscess at the level of the right hilus, with a fistulous communication into a large bronchus. He received parenteral penicillin with improvement and was discharged after six weeks, one month before his admission to this hospital. He continued to have fever and sweats. He raised large amounts of yellow, nonodorous sputum, and three days before admission the sputum became blood streaked for the first time. There had been marked dyspnea in the latter part of his illness. During the whole course he had lost 71 pounds.

On physical examination the fistula was draining small amounts of pus. There were dullness and diminished fremitus over the lower two thirds of the right chest anteriorly and posteriorly; the breath sounds were harsh and loud, and there were scattered fine rales over this area. The heart was slightly enlarged to the left, and there were numerous extrasystoles. The liver edge was 3 cm. below the costal margin. The appendectomy scar was healed.

The temperature was 101.5°F., the pulse 72, and the respirations 24. The blood pressure was 135 systolic, 65 diastolic.

The red-cell count was 3,590,000, with 10 gm. hemoglobin. The white-cell count was 30,000. The urine was normal. An x-ray film of the chest showed considerable radio-opaque material in a large cavity in the apex of the right lower lobe. There was hazy increased density throughout the rest of the lobe. The right half of the diaphragm was high and immobile.

A bilateral superficial femoral-vein ligation was performed on the second hospital day. Several unsuccessful attempts were made to aspirate the right pleural cavity. On the fifteenth hospital day, the liver was tender and there was spasm of the overlying rectus muscle. The total protein was 7.19 gm., with an albumin-globulin ratio of 0.84. The prothrombin time was 26 seconds (normal, 18 seconds). By x-ray examination the liver did not appear enlarged, and its upper outline was sharply defined. A gastrointestinal series failed to reveal any deformity of the stomach or duodenal loop. Abundant colon bacilli were cultured from the sputum. In the meantime the patient had had two attacks of substernal pain, one accompanied by dyspnea, and the temperature was spiking. Several abdominal films taken during thorotrast study showed an area of decreased density within the

lower liver margin centrally, where the dye was not absorbed. The liver abscess was drained on the thirty-third hospital day. Abundant colon bacilli were cultured from the fluid.

Postoperatively the patient had a low-grade fever and frequently required oxygen. On the eighth hospital day an x-ray film of the chest was considerably changed, showing multiple rounded indefinite areas of increased density throughout both lungs. The appearance of the right lower lobe was unchanged. Twenty days postoperatively, after the temperature had been normal for five days, the patient was discharged to a nursing home.

Second admission (two months later). After discharge the patient was bedridden and continued to have a cough, dyspnea and a chronic draining wound in the upper abdomen. Two days before readmission the cough increased and there was an exacerbation of fever, with chills and headache.

The patient was moribund on admission. The findings were altered from the previous ones only in degree of severity. Rales and rhonchi were heard over both lung fields. The process seen in the x-ray film of the chest had moderately increased. The foci of increased density were connected by a network of increased densities, which were somewhat beaded and irregularly distributed. Death occurred on the third hospital day.

Differential Diagnosis

Dr. Carroll C. Miller: This case presents the aspects of a picture puzzle of three pieces, which have to be fitted in proper relation to one another to explain the final outcome. These three pieces are the appendix abscess, resulting from a ruptured appendix, the lung abscesses and the liver abscess. It seems to me that the important feature is the sequence of events, provided that we can demonstrate a logical sequence. In the first place, I believe that there was nothing in this case that could be considered specific, that is, I do not believe that we are dealing with a specific infection. The mention of a liver abscess makes one think of amebic abscess or echinococcal disease of the liver, both of which can cause complications such as those that occurred in this case. In a man of this age we particularly think of carcinoma or malignant disease, and malignant disease in the lung or liver is frequently masked by infection. There is nothing in this man's history, however, that suggests malignant disease, although, of course, it may have been present. It is true that there was a loss of 71 pounds in weight, but after all, this man had a history, going back over a year, of chronic sepsis, and patients with this degree of chronic sepsis often lose weight. I believe that the weight loss can be explained on that basis.

Let us first consider the appendix abscess. These days we see, fortunately, few cases of late complications of appendix abscess. Whether this is due to a better handling of acute appendicitis and ruptured appendix or to chemotherapy, one cannot definitely say; it probably depends on both to a certain degree. The story of appendix abscess followed in a short space of time by chills, sweating, high fever and pain in the right lower chest make me believe that there was a spreading infection from the region of the appendix or cecum. Pylephlebitis, with a spread of the infection through the veins of the portal system into the liver and the subsequent formation of multiple liver abscesses, would explain the clinical signs and symptoms.

So far as the lung abscesses are concerned, there are two possible explanations. One, which is attractive, is that since this operation was for an acute ruptured appendix it undoubtedly was an emergency procedure. We do not know the details of the operation, what anesthesia was used or what complications were associated with the immediate operative or postoperative situation, but it is possible that material was aspirated from the stomach — that is, vomitus during induction or recovery from the anesthesia may have been aspirated into the lung, with the resultant formation of multiple lung abscesses. A good many cases of lung abscess come to this hospital in which this has been the story. Usually these abscesses start out as tiny foci of infection surrounding the aspirated material, many of them in various parts of the lungs but mostly in the lower lobes. These abscesses, particularly if they are peripheral, cause pleuritic reaction with effusion and eventual contamination of the pleural cavity, with the formation of empyema. So the empyema that was found and drained may be explained on the basis of an abscess going out from the central part of the lung to the periphery. That is one direction for the train of events. Another direction, which is not infrequent, is that in a cephalad direction: that is, the infection in the abdomen may have gone up under the diaphragm, perforated the diaphragm and caused an empyema, which in turn invaded the lung. This involvement of lung tissue from without is rarer, I should say, than involvement of the pleura from within the lung parenchyma. The liver abscess, as such, might have produced the same sequence of events, breaking through the diaphragm without peritoneal involvement and causing empyema and lung changes.

I should like to speculate a minute on the matter of chemotherapy, which has some bearing on this case. The patient was given sulfonamides and penicillin following the first bout of sepsis in the chest. So far as appendix abscess is concerned, we have had varying responses with the use of sulfonamides. Both sulfadiazine and penicillin have frequently seemed beneficial in cases of peritonitis from ruptured appendix. But these drugs are notorious, of course, for affecting the gram-positive organisms and not affecting the gram-negative ones, and that is borne out by the fact that in this case colon bacilli were cultured from two sources — the

sputum and the liver abscess. Apparently the use of sulfonamides inhibited the other organisms, so that there was a persistence of colon-bacillus infection in spite of the repeated use of penicillin and sulfonamides.

It is surprising that these abscesses of the lung were apparently not foul. This is relatively unusual, but on the basis of chemotherapeutic responses, we can assume that some of the organisms that ordinarily cause foul abscesses were killed by the chemotherapy. Blood streaking of the sputum is a finding that we see frequently in lung abscess. It is not necessarily a sign of infarction or of malignant disease, but we have to think about infarction and pulmonary emboli in relation to the other findings.

A point of interest to me is the reason for the superficial-vein ligation. In this hospital we are naturally highly sensitized to the matter of embolic catastrophes coming from the veins of the legs. I should be interested to know whether this ligation was done as a prophylactic measure or with the conviction that the episodes of pulmonary difficulty were secondary to thrombophlebitis of the legs. It seems that the latter was probably the case. We are not told whether clots were found in the veins, and no mention is made of the flow of blood from above. It would be of some interest in this case, because I suspect that there was a phlebitis in the abdomen above the point of ligation. In the early days of his illness this man had repeated episodes of chills, fever and chest pain. After he came to the hospital he again had episodes of pain, two of them substernal, accompanied by dyspnea, which to me suggest pulmonary infarction. Before we see the x-ray films I shall briefly mention the second admission, which simply showed an exacerbation of all the signs previously manifested, with, in addition, headache. This may have been simply a reflection of the sepsis and of the high temperature, but it is suggestive to me of the possibility of a metastatic brain abscess. The headache was not mentioned previously, and we know that the three most frequent causes of death in lung abscess are sepsis, hemorrhage and brain abscess.

May we see the x-ray films?

Dr. James R. Lingley: These are the first films and show the large area of density at the right base, which in the lateral view lies posteriorly and evidently represents encapsulated fluid. The appearance of the lipiodol suggests an alveolar distribution rather than lipiodol lying in a cavity. It was stated in the record that it communicated with the bronchus on the left side. In the lateral film there is a diffuse process, even at this time, of patchy areas of density distributed throughout the lung. This is the film taken five weeks later. There is a marked advance of the process throughout the left lung. The areas of involvement are small and circumscribed, yet it is a diffuse process involving the entire lung field.

Dr. Miller: Do you think that is a cavity?

Dr. Lingley: I cannot see anything that looks like a cavity except this area on the right.

Dr. Miller: It is possible that that might be an advance of the diffuse linear process seen in the previous film.

Dr. Lingley: Yes.

Dr. Miller: Have you the thorotrast films? They are of some interest.

Dr. Lingley: This film after thorotrast injection shows two areas of diminished radiance and rarefaction in the liver.

Dr. Miller: In recapitulation, I assume that this patient had a septic phlebitis arising in the region of the cecum and involving the iliac vein on that side and perhaps spreading up the inferior vena cava, although I find no sign of caval blockage in the picture that is described. I suppose that we must also predict a pylephlebitis to account for the liver abscess, because I do not believe that a process coming down from the lungs could have produced these multiple liver abscesses. So, although the temptation is great to ascribe the multiple lung abscesses to aspiration from the esophagus at the time of operation or shortly thereafter, I am a little more in favor of the picture's being explained on the basis of septic infarction of the lungs, arising from the right iliac vein, with metastases to the lung, and eventually from the lung to the brain to explain the possible brain abscess. The pylephlebitis may have followed an independent route, traveling through the portal system into the liver.

Clinical Diagnosis

Multiple abscesses of lung and liver.

Dr. Miller's Diagnoses

Thrombophlebitis, right iliac vein.
Septic lung infarcts, with abscess formation.
Pylephlebitis.
Liver abscesses.
Brain abscess?

Anatomical Diagnoses

Multiple abscesses, pericecal, hepatic and pulmonary.
Actinomycosis of lungs.
Operative wounds: appendectomy, old; drainage of empyema and lung, subdiaphragmatic and liver abscesses.
Arteriosclerosis, generalized.

Pathological Discussion

Dr. Tracy B. Mallory: At autopsy, we found abscesses in three areas. There was a persisting abscess in the region of the cecum that had not been entirely drained. There was a large abscess in the liver, which at the time of autopsy appeared as a single abscess, although it may well have been multilocular in the early stages, and there were multiple

abscesses throughout the lungs. We did not find a thrombophlebitis. The vein ligation was described as being prophylactic in character. Infection can, of course, spread by metastases through the venous system without producing progressive thrombosis. Although the clinical sequence seems to point to lung abscess and then liver abscess, I am inclined to think that the reverse order is more probable, the first process being in the liver and the second in the lungs.

You have already heard the bacteriologic report that colon bacilli were cultured from the sputum. When the sections came through we found microscopically an additional organism, which helped to explain the failure of response to chemotherapy. The abscesses in the lungs contained numerous actinomycotic granules. We did not find any in the liver, but I presume that the process there was of the same etiology.

We did not find any lesion in the brain.

CASE 32152

Presentation of Case

A twenty-one-year-old woman, a switchboard operator, entered the hospital complaining of severe headaches.

Four or five years before admission, she noticed the onset of diplopia. At first this condition was corrected by prismatic glasses; but it grew progressively worse, requiring frequent change of lenses, and one year before admission the diplopia persisted in spite of glasses. Approximately concomitant with the onset of the diplopia, the patient began to have "cramps" in the right hand. These were accompanied by a cold, numb feeling sometimes limited to the fingers and sometimes involving the entire hand. During these attacks the hand felt "frozen" in whatever position it had occupied at the onset; it did not tingle or feel numb. The right leg was never affected, and there was no disturbance of speech. One year before admission the patient began to have paroxysmal attacks of sharp stabbing pains distributed symmetrically over both cheek bones and sides of the nose but never involving the supraorbital regions, jaw, teeth or gums. These attacks occurred three to four times a day and lasted two to three minutes. They were accompanied by a cold, numb feeling having the same distribution as the pain but persisting constantly for about a week, during which the attacks of pain occurred. Then both pain and numbness subsided, to recur again for one-week periods once or twice a month. Six months before admission, the attacks of pain in the face subsided but there was an increase in the frequency and intensity of the mild headaches that she had been having occasionally ever since an illness, thought to have been undulant fever, at the age of seven. They became severe, stabbing and throbbing in character, extending diffusely over the head but occasionally becoming more marked over the temporal and occipital regions bilaterally. They often awakened her in the early morning, gradually fading in severity after two or three hours. The onset was usually accompanied by nausea, which was sometimes followed in about half an hour by vomiting. Occasionally the patient experienced a momentary "blackout" when she stood up during one of the severe headaches, but she never lost consciousness for more than a few seconds. She also complained of severe blurring of vision during the headaches. These attacks occurred about once or twice a week without apparent precipitating factors and did not respond to any form of treatment. During the six months before admission they did not increase in frequency but gradually became severer, lasting up to six hours and then gradually fading throughout the day.

The past history and family history were noncontributory.

Examination revealed a well developed and well nourished girl in no acute distress. There was papilledema of 2 or 3 diopters in the right eye, and 1 diopter in the left. The pupils were equal, being 4 mm. in diameter, and reacted normally to light and distance. There were convergent strabismus of the right eye and homonymous diplopia, persisting even on extreme lateral gaze to the left. There was no anosmia. The corneal reflexes and sensation of the face were normal. There was no facial asymmetry or weakness. An audiogram was normal. The palate and tongue were normal. There was no unsteadiness or weakness of the arms or legs. Sensation, including postural sense and stereognosis, was normal. The arm, knee and ankle jerks were brisk and bilaterally equal. There was no Hoffmann reflex. The plantar reflexes were normal.

The temperature was 99°F., the pulse 95, and the respirations 20. The blood pressure was 120 systolic, 75 diastolic.

The urine was normal. The red-cell count was 4,400,000, the white-cell count 8700, and the hemoglobin 80 per cent. The spinal fluid, which was under a pressure equivalent to 350 mm. of water, was xanthochromic and contained 30 red cells and no white cells per cubic millimeter. The total protein was 7 mg. per 100 cc., and the goldsol curve and Wassermann reaction were negative.

An electroencephalogram was abnormal, with slow activity increasingly prominent posteriorly. Although there was no well defined focus, the slow activity (4 to 5 seconds) was especially prominent in the left occipital and the right posterotemporal regions.

X-ray films of the skull showed unusual prominence of the convolutional markings. Only one posterior clinoid process, thought to be the right, was seen, and that seemed to have a blunted tip.

A ventriculogram showed both lateral ventricles well filled and normal in shape and position, but they appeared to be somewhat enlarged. No air was seen in the third ventricle, aqueduct or fourth ventricle. The foramen of Munro was visualized.

An operation was performed.

Differential Diagnosis

Dr. A. Price Heusner*: Before undertaking an analysis of this case I should like to ask two questions. Are the results of a visual-field examination available? Is the abstract correct in reporting that the spinal-fluid total protein was 7 mg. per 100 cc.?

Dr. Charles S. Kubik: That was ventricular fluid. The visual fields were normal except for large blind spots resulting from the papilledema.

Dr. Heusner: But nothing striking.

Dr. Kubik: No.

Dr. Heusner: We are asked to consider a twenty-one-year-old woman who was forced to enter the hospital because of increasing severity and frequency of headaches, which she had had for as many as twelve or fourteen years. It is quite difficult, I believe, to make sure whether these headaches were a part of the present illness or whether she had them from some other cause. In any event she had had headaches, and they finally forced her to the hospital, with nausea and vomiting. Five years before admission she noted the onset of what became a persistent diplopia, and at about the same time she began to suffer paroxysmal painful subjective sensations in the right hand. The latter apparently subsided after an unstated length of time, but some four years later they were followed by the development of similar paroxysmal sensations that occurred in a butterfly distribution over the face — in other words, they were bilateral.

The various examinations done at the time of admission give us unequivocal evidence of the existence of an elevated intracranial pressure: the disks were choked, the plain x-ray films showed convolutional atrophy, and the spinal tap revealed a pressure equivalent to 350 mm. of water. On the other hand, this bizarre symptomatology, extending back at least five and perhaps as many as fourteen years, did not leave in its wake anything that I can regard as a definitely localizing neurologic sign. On admission she had an internal strabismus of the right eye, which I take to be evidence of palsy of the right abducens nerve and adequate cause for the diplopia that she had experienced for several years. The note that the diplopia persisted even in extreme left lateral gaze permits of more than one neurologic interpretation. It might mean a beginning palsy of the left abducens nerve or a disturbance of the mechanism, situated high in the mid-brain, that regulates conjugate movements of the eyes. Because no one of these signs may be depended on for localization when it occurs in conjunction with an elevated intracranial pressure, it is not surprising that a ventriculogram was made. It would be helpful to see these films at this point and also the plain x-ray film of the skull, which was described as showing convolutional atrophy.

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Dr. James R. Lingley: These are the plain films showing the convolutional atrophy, as well as some thinning of the posterior clinoid process, but no definitely localizing signs.

Dr. Heusner: The sella is not ballooned?

Dr. Lingley: No; I should say that it is normal in size.

In the ventriculogram there is dilatation of both lateral ventricles, and you can trace air down into the anterior portion of the third ventricle. This small quantity of air in the anterior portion of the third ventricle outlines the anterior border of a mass that fills the posterior part of the ventricle. In the lateral view you can also see the mass projecting up into the floor of the lateral ventricles, more marked on the left side than on the right. The x-ray findings therefore indicate a mass filling the posterior portion of the third ventricle and projecting up into the floor of the left lateral ventricle.

Dr. Heusner: There is therefore objective evidence that air failed to reach the body and the posterior portion of the third ventricle. This suggests the presence of a mass in the back end of the third ventricle, but before accepting this as an absolute certainty it would be wise to inquire whether the suggested localization is clinically reasonable. Although it is true that the ventriculogram remains the neurosurgeon's most reliable single localizing adjunct, it is not a method without caprice. Occasionally, even in the most experienced hands, failure to visualize some portion of the ventricular system does not mean that that portion of the system is occupied by a pathologic mass. Let us, then, hearken back to the symptomatology and see if the localization suggested by this ventriculogram is clinically reasonable.

If we consider the over-all picture of a twenty-one-year-old woman with choked disks but without localizing signs, one of the first lesions we should think of is a neoplasm in the posterior fossa. We have, then, to ask ourselves whether it is more reasonable to expect a lesion in the posterior fossa or one in the posterior portion of the third ventricle, as suggested by the ventriculogram.

There are numerous reasons for accepting the ventriculographic localization in this case. The history and neurologic signs are not those of posterior-fossa disease. None of the cranial nerves in that region have suffered, and there are no signs or symptoms, such as ataxia, dysmetria and nystagmus, suggesting disorder of the cerebellum itself. Further, in posterior-fossa tumors the entire third ventricle shares in the hydrocephalic enlargement, a circumstance affording the best possible chance for filling that cavity with air.

On the positive side of the balance there are yet other reasons for thinking that the ventriculogram is a correct portrayal of the situation. The headache was periodic, but its periodicity was not without system. We note that the patient was frequently awakened by headache, but that on assuming the erect posture it subsided, at first within an hour or two and somewhat later in the disease only after five or six hours of being out of bed. This suggests that the headaches were due to intermittent obstructions to the flow of cerebrospinal fluid that were related to changes in posture. A mass situated in the third ventricle is certainly in a strategic location for producing such a ball-valve action on the circulating fluid. Supporting this as a possibility is the finding that the spinal fluid was yellow and contained a little blood.

DR. KUBIK: The lumbar puncture was done after the ventriculogram.

DR. HEUSNER: My abstract says that the spinal fluid was xanthochromic.

DR. KUBIK: I am sorry about that.

DR. HEUSNER: We cannot, then, call on xanthochromia as a means of supporting the possibility that something was protruding into the ventricular system, but that is a finding we have come to recognize as likely when some abnormal tissue is lying in a subependymal position or actually within a ventricle.

But there are other reasons for accepting the ventriculographic suggestion of a mass in the posterior portion of the third ventricle. If we are to explain the symptomatology on the basis of a single lesion, that lesion must be in close proximity to the central sensory components subserving both sides of the body, for although it is true that the pains began in the right hand, later in the disease they appeared bilaterally over the face. Certainly a mass in the third ventricle, the walls of which are composed in part of the thalami, important sensory substations, would be favorably situated to effect bilateral pain. I do not know how a lesion in that position actually produces pain, yet the clinical description of this patient's discomfort is to me not unlike what we sometimes see following a thrombosis involving the thalamus. For these several reasons, then, I am going to accept the ventriculogram as having demonstrated the presence of a space-occupying something in the posterior portion of the third ventricle.

This is a long history. What slowly growing things occur in this region? Accumulated experience shows that practically everything from granulomas and parasitic cysts through various tumors have been found in this location. But since we have nothing to suggest a granuloma or parasitic cyst, I am going to confine my comments to the reasonable statistical possibilities, namely, the tumors.

Of the tumors that come to occupy the third ventricle, the great majority arise outside the ventricle and eventually extend into it; the minority are primary intraventricular tumors. Of the tumors that arise outside and eventually gain access to this ventricle, the most frequent one is, I think, the Rathke-pouch tumor. In this case I do not believe that we need seriously consider such a lesion. The characteristic visual-field changes and suprasellar calcification are wanting, and what is more significant, the anterior portion of the third ventricle was visualized. Such a tumor should obstruct the anterior portion of this cavity before the posterior part is obliterated. On similar grounds I shall eliminate the occasional pituitary tumor that bursts its bounds, reaches the third ventricle and produces a hydrocephalus; the visual-field changes characteristic of that lesion are missing, the sella is not ballooned, and the anterior portion of the third ventricle was visualized. There is, however, a relatively frequent group of tumors arising in the pineal body near the back of the roof of the third ventricle. Such tumors not infrequently extend downward and come to fill a part or even all of this ventricle. I see no way of ruling out such a growth in this case, even though the patient did not show the large fixed pupils and impairment of upward gaze that are sometimes produced by such a mass pressing on the tegmentum of the midbrain.

There are two tumors that frequently arise within the third ventricle, if any third ventricular tumor may be said to be frequent — colloid cyst and papilloma of the choroid plexus. I see no way of definitely ruling out either of these. Yet, in the great majority of cases colloid cysts arise from the anterior portion of the roof of this ventricle,[1] and since that portion of this ventricle was visualized I should not expect this patient to harbor a colloid cyst. In other words, it seems as if this patient's mass originated more posteriorly than is usual for colloid cyst. Papilloma of the choroid plexus, on the other hand, is apt to arise from the posterior portion of the roof, and it is notorious that tumors arising in that region may go for a long time before they give rise to serious obstructive or neighborhood symptoms. So, it appears to me that the likeliest statistical possibilities in this case are pinealoma and papilloma of the choroid plexus.

The position of a mass occupying the third ventricle is of extreme importance to the surgeon, because if he can be certain of this his operative approach is likely to be successful. In a case of this type the surgeon is primarily interested in knowing whether the tumor is more accessible from the back or from the front of the ventricle. The air studies provide the answer: the tumor was more posterior than anterior.

My diagnoses are internal hydrocephalus, involving the lateral ventricles and the anterior portion of the third ventricle, and a tumor occupying the

posterior portion of the third ventricle — either a pinealoma or a papilloma of the choroid plexus.

DR. AUGUSTUS ROSE: How do you explain the diplopia?

DR. HEUSNER: To explain that I have to fall back on the experience of others. The tumors that occur in this region are rare. I have seen very few of them, but the symptoms in 2 of Dandy's[1] 15 cases of primary intraventricular tumor began with diplopia.

DR. ROSE: You think that it is a sixth-nerve palsy?

DR. HEUSNER: I do not know.

DR. ROSE: I do not know either.

DR. JAMES B. AYER: Does not a pineal tumor usually show in a ventriculogram by the time that it has done this much damage?

DR. HEUSNER: I believe so, and I also believe that Dr. Lingley pointed out a fullness extending up into the lateral ventricle in one of the projections.

I am glad that no one has asked me to explain the pain in this patient's arm or face, but again falling back on the experience of others, I find that similar pains have been observed, although not in

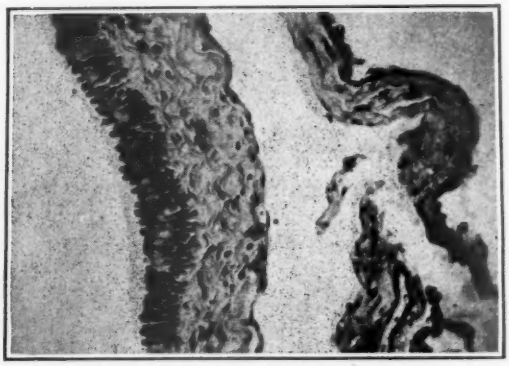

FIGURE 1.

a butterfly distribution. Those who have tried to formulate clinical syndromes of third ventricular disease have included among them a so-called "thalamic syndrome" resulting from tumors in this region and characterized by spontaneous, often paroxysmal, pain.[2, 3]

In my abstract the spinal fluid was said to be yellow. Had the tap been done before the ventriculogram, I should have favored a papilloma of the choroid plexus ahead of pinealoma, but without that, I assume that the spinal fluid was initially clear. On statistical grounds I guess that a growth arising in the pineal is the likelier.

DR. WILLIAM H. SWEET: The extreme duration of symptoms, as suggested by the convolutional atrophy of the skull, might also lead to the diagnostic possibility of an epidermoid tumor, since that is the slowest growing of the congenital-rest tumors that are seen in the third ventricle.

DR. KUBIK: Would you like to add anything more about the operative findings?

DR. SWEET: On the basis of the extreme duration of symptoms it appeared to me that a colloid cyst or an epidermoid tumor were the likeliest diagnoses. I searched a long time and finally found a small colloid cyst involving the third ventricle. I was not able to find any evidence that the lateral ventricle on either side had been encroached on by a space-taking mass and had a rather difficult task to get between the internal cerebral veins without hemorrhage. This was eventually achieved, and a small yellowish mass encountered, which was readily removable; it was not attached to any significant amount of the roof of the third ventricle, and after aspiration of the viscous fluid content, the collapsed capsule was readily lifted out.

DR. KUBIK: How large would you say that it was?

DR. SWEET: About 1.0 or 1.5 cm. in diameter.

DR. KUBIK: And the subsequent course?

DR. SWEET: The patient was apathetic postoperatively for a week or two. After two or three weeks she began to behave normally, and when heard from three months postoperatively she had no complaints and was leading an active life.

CLINICAL DIAGNOSIS

Colloid cyst of third ventricle?
Epidermoid tumor of third ventricle?

DR. HEUSNER'S DIAGNOSES

Internal hydrocephalus, involving lateral ventricles and anterior portion of third ventricle.
Tumor occupying posterior portion of third ventricle (? pinealoma or papilloma of choroid plexus).

ANATOMICAL DIAGNOSIS

Colloid cyst of third ventricle.

PATHOLOGICAL DISCUSSION

DR. KUBIK: The mass removed by Dr. Sweet, as he has already stated, was a colloid cyst of the third ventricle. A similar case has been reported.[4] The patient was a forty-nine-year-old woman who had been having headaches for one year and became comatose a week before entering the hospital. There were no focal signs. The cyst, measuring about 1 cm. in diameter, was attached to the roof of the third ventricle between the optic thalami. It is possible, I suppose, that its relation to the optic thalami accounted for pain or paresthesia, as suggested by Dr. Heusner. The section shows a thin cyst wall lined with columnar ciliated epithelium (Fig. 1). These cysts, which are benign and removable, are thought to originate from vestigial cells of the paraphysis.

REFERENCES

1. Dandy, W. E. *Benign Tumors in the Third Ventricle of the Brain: Diagnosis and treatment.* 189 pp. Springfield: Charles C Thomas 1933. Tables opposite p. 128 and p. 165.
2. Weisenburg, T. H. Tumors of third ventricle, with establishment of symptom-complex. *Brain* **33**:236-260, 1910.
3. Fulton, J. F., and Bailey, P. Tumors in region of third ventricle: their diagnosis and relation to pathological sleep. *J. Nerv. & Ment. Dis.* **69**:1-25, 145-164 and 261-277, 1929.
4. Shannon, E. W. Colloid cyst of third ventricle. *Arch. Neurol. & Psychiat.* **51**:570-572, 1944.

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THE INADEQUACIES OF MEDICAL CARE

I. General Considerations

What is wrong with the practice of medicine? As long ago as 1927 the Committee on the Costs of Medical Care was organized by a group of leading physicians, public-health authorities and economists to solve the problem — "the delivery of adequate scientific medical service to all the people, rich and poor, at a cost which can be reasonably met by them in their respective stations of life." Although extensive majority and minority reports were rendered at that time, matters have little improved, and the point has finally been reached at which compulsory health insurance is advocated by President Truman, certain congressmen and others as the *only* means by which the health of the Nation can be assured. Such a scheme or any other plan for distributing the costs of medical care concerns merely one of the many factors that contribute to the health of the Nation. Similarly, the art and the science of medicine, which admittedly are developed in the United States to a degree that is unsurpassed in any other country, comprise another single factor. In other words, adequate and proper medical care for all largely depends on various socioeconomic factors with which the majority of physicians have had little experience and over which they have little control.

In any broad concept of matters pertaining to national health many factors have an equal, if not greater, influence than has medical care. These include good wages, a high standard of living, adequate housing and transportation facilities and a public well educated concerning proper diets, methods of preventive medicine and means of obtaining medical care. Although the lack of one or more of these factors renders the success of any system of medical care questionable, if not impossible, they have received little consideration by those clamoring for compulsory health insurance. Such matters, of course, are not true responsibilities of the medical profession, except for the fact that physicians might well play more of a role than they have in the past in matters of lay education. On the other hand, many inadequacies pertaining directly or indirectly to medical care exist, and the refusal to recognize and admit these faults has done much to lead to the position in which, unfortunately, the medical profession now finds itself.

In the last fifty years, the science of medicine has made tremendous progress, possibly at a sacrifice to the art. The "family doctor" as he existed in the nineteenth century is a person of the past, since it has become impossible for any one physician to care properly for *all* the aches and pains of his clientele. Although the man in general practice will undoubtedly continue to comprise the bulwark of the medical profession, being just as essential to it as was the infantry to the modern army, this advance in the science of medicine frequently demands elaborate diagnostic aids and the advice and technical skill of the so-called "specialist." In cases of serious illness, it has put a premium on hospitaliza-

tion and all that goes with it. Many persons, however, are unable to obtain admission to a hospital, either because the facility is nonexistent or because beds are unavailable, and combined with this lack or scarcity of hospital facilities is the apparent inability of the medical profession to adapt itself to the changing practice of medicine, either wishfully or because of ignorance of the basic factors that are involved.

The subsequent editorials of this series will point out what appear to be the present-day inadequacies of medical care — as they concern its distribution and cost, its quality and the various adjuncts that are closely associated with, if not essential to, its successful accomplishment — and to hazard a guess concerning what must be done if the health of the Nation is to be improved.

PENICILLIN AND SYPHILIS: A SECOND WARNING

In a previous editorial in the *Journal*,[1] attention was called to the known fact that syphilis may lie dormant for long periods and later produce clinical effects or be transmitted to a second generation. Emphasis was laid on the importance of serologic follow-ups over a period of years in all persons who have been treated with penicillin for syphilis so that the exact potentialities and end-results of this method may be fully determined. In particular, returning veterans who had been treated for syphilis were mentioned as requiring this follow-up, because in many of them, early in the experimental period of this form of therapy, relatively small doses of penicillin were used. The same, however, may also be said of civilians — including those treated more recently with various combinations of penicillin with arsenicals and bismuths.

Of equal and perhaps greater importance is the need to search for serologic and clinical evidence of syphilis in patients who have been treated with penicillin for gonorrhea. The fact that gonorrhea and syphilis are frequently acquired at the same time but have different incubation periods, as well as the nature of the effect of small doses of penicillin on the early manifestations of these two infections, renders the masking of the early manifestations of syphilis a definite possibility. Thus, the incubation period of gonorrhea is usually three to five days and only rarely more than a week, and its manifestations are usually obvious enough to the patient so that medical treatment is sought. As little as 100,000 units given in four or five intramuscular doses over a period of six to nine hours or a single dose of 200,000 or 300,000 units in a beeswax-peanut oil mixture produces complete cures in the great majority of cases. The incubation period of syphilis, on the other hand, is usually about three or four weeks and may be much longer. Treatment for gonorrhea may, therefore, be given some time before the primary syphilitic lesion appears.

Leifer and Martin[2] have recently reported on 15 cases of simultaneous infections from their own experience and from the literature. They very properly point out that for various reasons penicillin treatment of gonorrhea may be undertaken when primary syphilis is already present but overlooked or ignored. For example, a primary lesion may be missed because of its unusual location, it may be concealed by inflammatory phimosis, it may be regarded as an unimportant lesion or the diagnosis may seemingly be excluded by one or more negative dark-field examinations or by negative serologic tests for syphilis. In the earliest studies on the penicillin treatment of syphilis it was shown by Mahoney, Arnold and Harris[3] that the primary lesion undergoes rapid changes within a few hours after treatment is started. With doses of 25,000 units every four hours, spirochetes disappear from the lesion, as shown by dark-field preparations, within sixteen hours or even earlier. There may also be clinical symptoms within the first eight hours that suggest a therapeutic effect. These include fever, pain at the site of the lesion and enlargement or tenderness of the regional lymph nodes; occasionally a brief rash resembling that of secondary syphilis appears.

Leifer and Martin also point out that the small doses of penicillin that are used in the treatment of gonorrhea may conceivably bring about a cure of a coincidental early syphilitic infection in rare cases. It is likelier, however, that a chancre will appear perhaps after a prolonged period of incubation. It

may be expected that treatment in the primary stages of syphilis will heal the lesion prematurely, after which it may either recur, the disease may be rendered asymptomatic or a secondary eruption may appear. In addition, a Herxheimer reaction may be evoked in the course of treatment, either at that time or on some subsequent occasion when penicillin is given. In the cases that they studied, the diagnosis of syphilis was proved in every instance either by demonstration of spirochetes in the lesions or by repeated positive serologic tests in less than ninety days after penicillin had been administered. On the basis of their observations these authors recommend that all patients treated with small doses of penicillin for gonorrhea should have clinical and serologic observations for a period of three months.

Another interesting feature of penicillin treatment may be anticipated and has probably already been observed by some physicians. Many persons with latent or manifest syphilis undoubtedly receive penicillin therapy for other conditions, the drug being given for a brief course while the patient is acutely ill or in larger doses over longer periods for the treatment of such conditions as septic wounds, osteomyelitis and subacute bacterial endocarditis. In any event, an early febrile reaction may occur during the first few hours of penicillin treatment, and this may be accompanied by symptoms of various severity which may be easily overlooked or misinterpreted. A rise in fever, a chill or a headache may be considered as a phase of the infection that is being treated, and a rash may be considered as a sensitivity reaction to penicillin, whereas actually these signs and symptoms are manifestations of a Herxheimer reaction. Physicians should be on the alert for these types of reactions and should obtain blood for serologic tests for syphilis at such a time. Positive results may be reported in occasional cases in which previous tests have been negative or in which the diagnosis of syphilis has not otherwise been suspected.

References

1. Editorial. Penicillin and syphilis: a warning. *New Eng. J. Med.* **233**: 53, 1945.
2. Leifer, W., and Martin, S. P. Effect of penicillin on course of early syphilis. *J. A. M. A.* **130**:202-205, 1946.
3. Mahoney, J. F., Arnold, R. C., and Harris, A. Penicillin treatment of early syphilis: preliminary report. *J. Ven. Dis. Inform.* **24**: 355-357, 1943.

OBITUARY

SAMUEL BAYARD WOODWARD

1853–1946

Samuel Bayard Woodward, the son of Samuel Woodward, a merchant of Worcester, Massachusetts, and his wife, Elizabeth Rogers Treadwell Woodward, was born August 24, 1853. On his father's side he was a descendant of Dr. Henry Woodward, who settled in Dorchester in 1653 and carried on the practice of medicine there. On his mother's side, the members of Thomas Treadwell's family were fellow passengers on the ship with Dr. Henry Woodward. They settled in Ipswich.

Another Woodward, Dr. Samuel, lived in Torrington, Connecticut, and Dr. Samuel Bayard Woodward I, grandfather of the subject of this obituary, settled in Weathersfield, Connecticut, later moving to Worcester, Massachusetts. He was the first superintendent of the Worcester Lunatic Hospital.

His grandson, after an adequate premedical preparation, including an A.B. degree from Harvard College, entered Harvard Medical School and graduated as one of the honor men of his class. He then was appointed house officer at the Boston City Hospital, where he won the confidence of the visiting staff and his associates. Having decided to prepare himself for the greatest possible usefulness in dealing with the problems of disease, he studied abroad for three years, visiting the hospitals of Dublin, London, Heidelberg, Vienna, Strasbourg and Paris.

On his return to his native city he immediately sought the advice of the older doctors and decided to settle in Worcester. He joined the Massachusetts Medical Society in 1877 and immediately became active in the affairs of the Worcester District Medical Society, being elected treasurer in 1886 and president in 1902. His election to the Council of the state society followed the next year. These positions, together with his activity on committees, were preludes to his election as president of the state society in 1916. Since his administration of the affairs of the Society was noteworthy, he was asked to accept the honor of a three-year term, a distinction not previously conferred since 1862.

His service during these years was especially arduous because of the threat leading up to and the declaration of war in 1917, which necessitated raising the required medical personnel for the Army and Navy, together with frequent meetings of legislative committees. Health insurance was being considered throughout the country at that time, and the Council appointed a committee to study the subject; but no reports were rendered and many years elapsed before this matter was brought up

again and a successful plan adopted. Workmen's compensation also engaged the attention of the Society during the war years, and the law relating to this important economic matter was put into workable form after much study and many amendments.

By reason of Dr. Woodward's reputation for executive ability, he was requested by many financial institutions to help carry on the work of their corporations. In 1885 he united with others in the incorporation of one of the large savings banks of the state, later becoming its president. The success of this bank led to the development of a national bank and other savings banks in Worcester, with his name on the list of officers.

These activities led in turn to other important positions in a great variety of active corporations engaged in business, philanthropy, education and sociologic activities to the extent of thirty or more units to which he gave his time in meetings and important decisions, a most remarkable combination of responsibilities for a busy doctor to assume. His friends became concerned because of the likelihood of his inability to carry on so much without a breakdown, but his physical and mental energy seemed adequate for the demands made on his time and strength, and he continued this routine of attending innumerable meetings throughout the thirty or more years of his professional work and never relegated his medical interests to second place.

In addition to his hospital appointments, he especially enjoyed the position of trustee of the Worcester State Hospital, to which he gave much time, only to resign in 1914 under protest because of the change made to a paid board of three in place of the former unpaid board. He was able a few years later to have this serious mistake corrected.

His long contest before legislative committees to prevent the repeal of the law requiring the vaccination of school children covered many successive years. His voluminous papers, copies of addresses and many letters used in his arguments during this campaign were deposited in the Boston Medical Library several years ago.

These brief references to the work done and positions occupied by this public-spirited physician, who served his community, as well as his state, in many effective ways set a pattern of living that might well inspire others of equal strength and understanding to plan a routine of equal service to those who are in need of assistance and guidance, especially those who are victims of misfortune.

Two legacies in Dr. Woodward's will demonstrate his affirmation that medicine was his major interest. One is a large bequest to the Worcester District Medical Society for the creation or acquisition of a building in which meetings can be held and all the functions of the society can be efficiently carried on. The second is a grant to the Massachusetts Medical Society of an amount to augment the provisions of the Cotting Fund, established by a former active and devoted member for the purpose of entertaining those members of the Council in attendance at its meetings, a wise and effective provision designed to promote harmony among the members.

Dr. Woodward has in a great variety of ways left with us a memory of his ideals and successes that should encourage emulation.

W. P. B.

MASSACHUSETTS MEDICAL SOCIETY

DEATHS

COUSENS — Nicholas W. Cousens, M.D., of Waltham, died March 19. He was in his eighty-second year.

Dr. Cousens received his degree from the Medical Faculty of Trinity University, Toronto, in 1891.

His widow and three children survive.

SULLIVAN — Cornelius A. Sullivan, M.D., of Braintree, died March 19. He was in his sixty-seventh year.

Dr. Sullivan received his degree from Tufts College Medical School in 1904. He was past president of Norfolk South District Medical Society, medical consultant, Norfolk County Hospital, staff member of South Shore Hospital, South Weymouth, and Braintree school physician for many years. He had been associate medical examiner for Norfolk County for twenty-two years. He was a fellow of the American Medical Association.

His widow, a son and four daughters survive.

NOTICES

ANNOUNCEMENTS

Dr. Julius Abramson announces his return from military service and the opening of his office for the practice of cardiology at 127 Bay State Road, Boston.

Drs. J. Englebert Dunphy and Stanley O. Hoerr announce the resumption of the practice of surgery at 721 Huntington Avenue, Boston, and 1101 Beacon Street, Brookline.

Dr. George H. R. Gosman announces that Dr. Joseph W. Tiede, having recently returned from active service, will be associated with him in practice at 121 Water Street, Boston.

Dr. Henry W. Hudson announces his return to the practice of surgery with offices at 1672 Beacon Street, Waban, and 1101 Beacon Street, Brookline.

Dr. Bernard M. Jacobson has returned from Naval duty and will resume the practice of internal medicine at 276 Commonwealth Avenue, Boston.

Dr. Elihu I. Lewis, having been released from active duty with the United States Navy, announces his return to the practice of internal medicine at 510 Commonwealth Avenue, Boston.

SUFFOLK DISTRICT MEDICAL SOCIETY

The spring dinner meeting of the Suffolk District Medical Society will be held at the Harvard Club of Boston on Saturday, April 27, at 7 p.m. Representative Edith Nourse Rogers will speak on the subject "Medical Legislation in Congress for Veterans" and Dr. J. C. Harding, assistant medical director for the auxiliary services of the Veterans Administration, will speak on "Highlights in Problems Relating to the Civilian Doctor and the Medical Care of Veterans."

Members and their wives are cordially invited to attend. The tickets which are $3.00 each must be purchased in advance by sending cash or check to the treasurer, Dr. Richard S. Eustis, 319 Longwood Avenue, Boston 15, before April 20. After this date, other members of the Massachusetts Medical Society and their wives may apply.

(*Notices continued on page xix*)